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Issued February 1932
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MARKET DISEASES OF FRUITS AND VEGETABLES

POTATOES

By

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INTRODUCTION

BOTANICAL RELATIONS

The Solanaceae, or nightshade family, include four species whose vegetative organs or fruits are used as fresh vegetables. The most

[1] This publication is the first in a series designed to aid in the recognition and identification of pathological conditions of economic importance affecting fruits and vegetables in the channels of marketing, with a view to facilitating the market inspection of those food products and reducing and preventing losses from such conditions. The series represents an extended revision and elaboration, with the addition of illustrations, of a preliminary publication entitled "Handbook of the Diseases of Vegetables Occurring Under Market, Storage, and Transit Conditions," by George K. K. Link and Max W. Gardner, which was published by the Department of Agriculture in 1919 especially for the use of the food-products inspectors of the Bureau of Agricultural Economics and was never distributed to the public. In addition to his part in the authorship of the preliminary edition, Doctor Gardner participated in a portion of the revision embodied in the present publication. The material is organized on the basis of the botanical families to which the plants belong, but no botanical system is followed in arranging these families. Practical considerations make it desirable to issue the material in separate sections arranged somewhat in order of the economic importance of the crops. The Host Index of the Fungi of North America, by A. B. Seymour, 1929, is used as a guide to the nomenclature of causal fungi and to the names of authorities therefor. The colored plates are reproduced from colored photographs prepared through the collaboration of Webster Bros., Chicago, Ill., under the direction of George K. K. Link, Max W. Gardner, and Glen B. Ramsey.

important of these, the potato, *Solanum tuberosum* L., is also the most important of all vegetable crops. The others, in the order of their importance, are the tomato, *Lycopersicon esculentum* Mill.; the pepper, *Capsicum annuum* L.; and the eggplant, *Solanum melongena* L. All of these are natives of tropical regions. The eggplant, pepper, and tomato are warm-season plants, while the potato is favored by relatively cool growing conditions.

THE POTATO OF THE MARKET

The potato of the market is the tuber of the herbaceous annual *Solanum tuberosum*, which produces lateral underground stems, the stolons. The tuber is a swelling arising at the end of these stolons, and therefore is a vegetative organ in that it is a fleshy underground stem.

The so-called early potatoes of the market, which come mainly from the Southern States, are tubers that are harvested before the vines have matured and are marketed before they have attained full size and maturity. As a result they have a poorly developed, thin, and tender cork layer, which is easily rubbed off, exposing the tissues beneath, with resultant water loss, discoloration, and increased susceptibility to decay. Because of the poor development of the cork layer and the high water content of the tubers, new potatoes lose water rapidly and excessively, so that they easily lose the firmness which is a desirable market quality.

The so-called late or main-crop potatoes, which come from the Middle Atlantic and Northern States, are tubers which either have been allowed to mature naturally with the maturing vine or have been allowed to lie in the soil for 10 to 14 days to give opportunity for cork development in the case of those vines which have been killed prematurely by frost, destructive animals, or parasites or have been killed intentionally with chemicals to hasten maturity of the tubers.

Such tubers have a well-developed thick and tough cork layer and consequently are suitable for storage. If they are stored at relative humidities of 65 to 95 per cent and at temperatures between 36° and 41° F., there is a relatively short period of high initial water loss, followed by moderate shrinkage during the remainder of the storage period due largely to water loss and to a much lesser extent to digestion and respiration.

Firmness in a potato tuber, therefore, depends to a great extent upon the stage of maturity of the tuber at the time of harvest, the conditions to which it is exposed after harvest and in storage, and the length of the period that has elapsed between harvest and the examination of the tuber.

Texture and color of the cork also determine the market qualities of tubers. In netted varieties good development of netting, and in red varieties good development of color, constitute desirable market qualities.

Different varieties of potatoes are adapted to various culinary uses. Once a potato variety has established itself in the market because of its flavor and culinary qualities, its marketability is determined by many qualities, most of which are considered criteria in determining the grade of tubers. Among these are minimum

sizes for round and long varieties, and absence of anomalies and of pathological conditions or of their effects, which may be directly or indirectly induced by heredity, by mechanical, physical, or chemical factors, by animal (especially insect) depredators, or by parasitic plants or animals.

PATHOLOGICAL CONDITIONS AFFECTING MARKETABILITY

Certain pathological conditions of the vines affect the tubers only indirectly, but result in fewer marketable tubers because of a smaller setting or the development of small, poorly shaped, poorly colored, or flabby tubers. The most important of these pathological conditions are: Anthracnose (*Colletotrichum atramentarium* (B. and Br.) Taubenhaus); Cercospora leaf spot (*Cercospora concors* (Casp.) Sacc.); insect injuries of various kinds induced by the potato leaf hopper (*Empoasca fabae* Harris), the tarnished plant bug (*Lygus pratensis* L.), the Colorado potato beetle (*Leptinotarsa decemlineata* Say), blister beetles (*Epicauta* spp.), the potato aphid (*Illinoia solanifolii* Ashm.), the spinach aphid (*Myzus persicae* Sulz.), the potato psylla (*Paratrioza cockerelli* Sulc.), the potato stalk borer (*Trichobaris trinotata* Say), and the common stalk borer (*Papaipema nebris nitela* Guence); stalk rot (*Sclerotinia sclerotiorum* (Lib.) Massee); tip burn; and the virus disease, mosaic. Tubers from vines affected with mosaic are wholly unsuited for seed purposes.

Certain environmental factors, animal depredators, and parasitic animals and plants not only kill or weaken the vines, thus affecting production directly and the marketability of tubers indirectly, but they also affect tubers directly, destroying them completely, thus reducing production, or lowering their quality so that they become unmarketable. This group, to which belong some of the most serious field diseases of potatoes, includes: Blackleg (*Bacillus phytophthorus* Appel)[2] and *Bacterium solanacearum* (E. F. Smith) E. F. Smith;[3] drought and heat injury; Alternaria tuber rot or early blight (*Alternaria solani* (Ell. and Martin) Jones and Grout); flea-beetle injury (*Epitrix cucumeris* Harris and *E. subcrinita* Leconte); freezing injury; Fusarium tuber rots (*Fusarium* spp.); Fusarium wilts (*Fusarium* spp.); late blight or tuber rot (*Phytophthora infestans* (Mont.) DBy.); potato wart (*Synchytrium endobioticum* (Schilbersky) Percival); powdery scab (*Spongospora subterranea* (Wallr.) Lagerh.); root knot (*Caconema radicicola* (Greef) Cobb); Rhizoctonia scab, stem rot, and root rot (*Rhizoctonia solani* Kühn);[4] Sclerotium rot (*Sclerotium rolfsii* Sacc.); southern bacterial wilt (*Bacterium solanacearum* (E. F. Smith) E. F. Smith; tuber-moth injury (*Phthorimoea opercuelella* Zeller); Verticillium wilt (*Verticillium albo-atrum* Reinke and Berth.); and violet root rot (*Rhizoctonia crocorum* (P. ex Fr.). Among the virus diseases, leaf roll (net necrosis) and spindle tuber are the only ones that produce direct effects in tubers and are of market significance in table and seed stocks.

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[2] Syn., *Erwinia atroseptica* (Van Hall) Bergey et al.
[3] Syn., *Phytomonas solanaceara* (E. F. Smith) Bergey et al.
[4] Syn., *Corticium vagum* Berk. and Curt.

Certain pathological conditions are restricted entirely to the tubers and are induced either in the field alone or both in the field and after harvest, especially in storage and in transit. This group includes: Armillaria rot (*Armillaria mellea* (Vahl) ex. Fr.); bacterial soft rots (*Bacillus carotovorus* Jones,[5] *B. aroideae* Town.,[6] and *B. mesentericus* Trevisan); black-heart; chemical injury; drought and heat necrosis; enlarged lenticels; fowl injury; greening; growth cracks; hollow heart; insect injuries of various kinds caused by the potato-scab gnat (*Pnyxia scabiei* Hopt.), the seed-corn maggot (*Hylemyia cillicrura* Rondani), the potato seed-piece maggot (*H. trichodactyla* Rondani), white grubs (*Phyllophaga* spp.), and wireworms (several species of the family Elateridae); internal brown spot; leak (*Pythium* spp.); pink rot (*Phytophthora* spp.); Rhizopus rot (*Rhizopus* spp.); rodent injury; scab (*Actinomyces scabies* (Thax.) Güssow); second growth; silver scurf (*Spondylocladium atrovirens* Harz.); and Stysanus rot (*Stysanus stemonitis* Cda.).

Those pathological conditions of vines which do not produce definite anomalies or lesions in tubers are not discussed here.[7]

Plates 1 and 2 afford an example of losses caused by diseases of potatoes in transit to market.

ALTERNARIA TUBER ROT (EARLY BLIGHT)

(*Alternaria solani* (Ell. and Martin) Jones and Grout)[8]

OCCURRENCE, SYMPTOMS, AND EFFECTS

It appears now, contrary to views previously held, that *Alternaria solani* is able to attack tubers in transit and in storage, and consequently is of direct importance in affecting their marketability. So far, the greatest damage has been observed in shipments from Florida, Maine, and Wisconsin, especially in the Spaulding Rose, Early Rose, Green Mountain, Irish Cobbler, and Bliss Triumph varieties.

In addition to the vines and tubers of potato, the fungus attacks other members of the nightshade family, especially eggplant and tomato. In the eggplant, as in the potato, the damage done to the edible part of the plant is for the most part indirect and due to premature death of the foliage, which causes decreased yields. The fungus *Alternaria solani*, like other Alternaria species, attacks plant tissues most readily in the field and after harvest, when they have passed their stage of greatest vigor, especially when they are exposed to unfavorable environmental factors. Potato tubers become infected at harvesting time, and the development of lesions takes place when the tubers are in transit or in storage.

Lesions on the potato tubers involve the cork and cortical parenchyma and are of various sizes and shapes. Generally they are sunken

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[5] Syn., *Erwinia carotovora* (Jones) Holland.

[6] Syn., *Erwinia aroideae* (Town.) Holland.

[7] For information on diseases of potatoes not encountered on the market, but often affecting marketability, the following list of literature citations should be consulted: General diseases—*3, 4, 8, 12, 13, 26, 31, 34, 50, 66, 68, 73, 77, 81, 89, 101, 112, 123, 128, 133, 151, 175, 179;* degeneration diseases—*39, 40, 54, 65, 146, 159, 160;* leaf roll—*1, 2, 6, 20, 41, 67, 119, 122, 127, 192;* mosaic—*30, 71, 131, 138, 157, 161, 172, 178.* Other citations are given at the end of each section relating to a particular disease or condition. Italic numbers in this footnote and those in parentheses in the text refer to Literature Cited, p. 53.

[8] Throughout this publication the scientific name of the organism inducing a disease is shown in parentheses immediately following the heading giving the name of the disease. For a fuller statement regarding the rôle of these organisms, see footnote 9, p. 5.

Section of a railroad yard for fresh vegetables and fruits in Chicago, after reconditioning a shipment of southern potatoes severely affected with bacterial soft rot. To render it marketable, the entire lot had to be resorted and the sound tubers put into new sacks, entailing additional charges for labor and material. The piles of discards are being sorted by the poor of the city in search of edible remnants

Interior of one of the cars of the train shown in Plate 1, showing the discarded potatoes left in the car after sorting out the salable stock from a shipment severely affected with bacterial soft rot and scald

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A. Blackleg, eastern type
B, C. Alternaria tuber rot
D. Blackleg, western type
E. Silver scurf

The abnormally pointed type of tuber shown as D is often found in spindle tuber

with regular raised borders, and the cork has a purplish-brown metallic hue. Most lesions are superficial and vary from one-fourth to 1 inch in diameter. (Pl. 3, B.) Externally these spots may appear very similar to those produced by late blight, but internally the lesions are more shallow and are sharply set off from the healthy tissues by a layer of wound cork. This layer of cork makes it possible for portions of the decayed tissue to be lifted out with the point of a knife. A yellowish discoloration of the flesh is sometimes produced just beyond the edge of the lesion, but the decay does not feather out irregularly into the healthy tissue as is common in late blight. (Pl. 3, C.)

The symptoms on the foliage of potato, eggplant, and tomato are very similar. In fact, there is great uniformity in the lesions caused by any Alternaria species on living foliage. Lesions on the leaves are brown to black and usually show a series of concentric rings which produce a target-board effect. There is often a narrow marginal faded zone. The spots are usually oval in shape. On vigorous leaves, however, they may remain small and angular because of limitation by the veins. As the leaf weakens or dies, the spots enlarge. Under conditions favorable for the fungus the lower leaves of plants gradually die and dry up until only a few green spotted leaves remain at the top of the plant. In severe cases lesions develop on the petioles and upper stems of the plant. Sometimes the olivaceous to black fungus, which comes to the surface of the lesions, becomes abundant enough so that it can be seen.

CAUSAL FACTORS [9]

The fungus *Alternaria solani*, which induces this disease, overwinters in the form of mycelium and conidia in plant trash and possibly on tubers as well. In the spring it produces a new crop

[9] In this publication the section Causal Factors under each disease comprises a statement of the complex of circumstances that determine the occurrence of the pathological phenomena under consideration; in other words, a discussion of all known antecedents of the consequences which are recognized as the disease. It is common usage among American plant pathologists to refer to the fungus or bacterium which enters into the condition complex of a pathological phenomenon as "the cause" of the disease. It should be borne in mind, however, that even in a disease induced by a foreign organism many other factors usually determine the occurrence of the disease. Among these are the conditions that govern the occurrence, abundance, and virulence of the foreign organism, foremost among which are (1) such external nonliving environmental factors as temperature, moisture, humidity, and food supply, and (2) the genetic constitution and other internal environmental factors of the organism itself. In addition, occurrence of the disease is conditioned by those factors which determine the resistance and susceptibility of the affected plant, namely, (1) the genetic constitution and other internal environmental factors, such as the age of the plant, and (2) the reaction of the plant during growth, harvest, transit, and storage, to external nonliving environmental factors. Among these the most important are soil, weather, temperature, humidity, chemicals, composition of the surrounding atmosphere, and agents that cause injuries of any sort, especially those that break the protective tissues of the plant. For those pathological phenomena in which no foreign organism enters as a part of the causal complex, either the internal factors of the plant itself or the external nonliving environmental factors, alone or in combination, constitute the sole determining antecedents of the disease. The external nonliving environmental factors play a very important rôle in the pathological phenomena of plants during the growing, harvesting, transporting, and storage of the crop. As in diseases excited by foreign organisms, the significant external nonliving environmental conditions consist of physical, chemical, and mechanical factors, such as temperature, moisture, humidity, chemicals, composition of the atmosphere, and the like. When a specific foreign body, in the form of an organism, such as a bacterium, fungus, nematode, or insect, is the condition or agent which, by virtue of a precedent condition complex, is able to induce or incite a disease, the authors prefer to designate this specific type of disease factor or condition by the term "excitant" rather than by the expressions "the cause" or "causal organism," and to say that it "incites" or "excites" rather than that it "causes" the disease. In conformity with past usage in publications of the United States Department of Agriculture, however, the term "causal organism" is used rather than "excitant" in the body of this publication.

of conidia, which are easily dislodged and readily disseminated by wind, rain, and insects. Frequent rains and dews are necessary for spore production. The germinating conidia enter the tissues by germ tubes through the stomata or directly through the epidermis. The fungus produces spores when the spots are one-eighth to one-sixth inch in diameter, and a single spot may produce 1,500 to 5,000 spores in one season. On artificial media the fungus grows best, and its spores germinate most abundantly at temperatures between 77° and 85° F. There is no growth below 35° or above 112°.

Climate and soil exert a great influence upon development of the disease in the field. The most favorable conditions for the fungus and the disease are periods of high temperatures alternating with moist periods, in combination with a weakened condition of the plant. The fungus may attack both young and old tubers, the lesions becoming visible within about 10 days. Development of decay is very slow at 41° to 45°, most rapid at about 59°, and practically inhibited at 77°.

CONTROL MEASURES

In view of the fact that the parasite overwinters in plant débris, sanitary measures are recommended. These are crop rotation and destruction of the tops of affected vines. It is desirable not to pile vines on or near the tubers at digging time. Spraying with a 5–5–50 Bordeaux mixture, if done thoroughly and regularly (weekly), gives good control of the disease. (*38*, *55*, *56*, *143*, *189*.)

ARMILLARIA ROT

(*Armillaria mellea* (Vahl) ex Fr.)

Armillaria rot has been reported only from the State of Washington, where it is a factor only when potatoes are planted in newly cleared land that is rich in humus.

The fungus *Armillaria mellea*, which induces this rot, furnishes the diagnostic characters of the disease. It forms structures known as rhizomorphs, from which mushroomlike fruiting bodies arise. These, because of their color, have given rise to the name "honey fungus." The chocolate-colored rhizomorphs grow about the roots and penetrate the corky layer of the tuber and, attaching themselves, draw food material from the tuber until it becomes a shriveled mass.

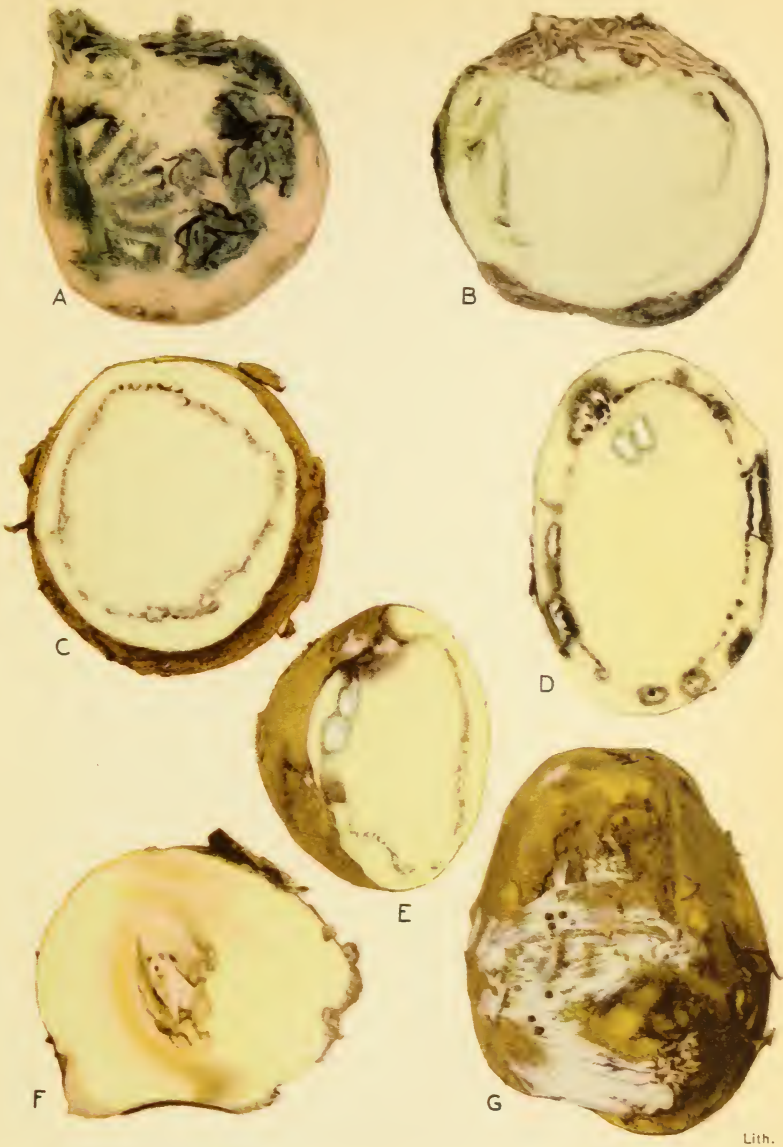
The disease can be avoided by planting corn or summer crops instead of potatoes in newly cleared humus soil until the fungus is starved out. Host resistance appears to be due to physiological antagonism between host and parasite rather than to structural differences. (*11*, *177*.)

BACTERIAL SOFT ROTS (SLIMY SOFT ROT)

(*Bacillus carotovorus* L. R. Jones; *B. aroideae* Towns.; *B. mesentericus* Trevisan)

OCCURRENCE, SYMPTOMS, AND EFFECTS

The term bacterial soft rot is applied to a group of bacterial rots of potato tuber aside from the blackleg rot (see Blackleg), from which



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A, B. Bacterial soft rot or slimy soft rot

C, D, E. Brown rot or southern bacterial wilt

F, G. Sclerotium rot

these can not always be differentiated with certainty unless cultural studies are made. This group of rots is also called slimy soft rot.

The decay is typically white to cream colored, with browning at the margins at times. Sometimes, and in some varieties, especially after exposure to air and light, it may become brown to black. The affected tissues are soft and elastic, but usually not decidedly watery. In some types of decay the affected tissues are viscous so that they can be pulled like taffy.[10] The boundary between the soft, disintegrated and the sound, firm tissues is very sharp, and often the outer portions of the tuber are left intact while the inner are completely destroyed. As the soft tissues dry they become slimy, and finally, when completely dry, they may be reduced to mere chalky white masses. (Pl. 4, A, B.)

In nature the bacterial soft rots are often attended by foul odors caused by other than the primary causal organisms. With pure-culture inoculations no odors, or only slight ones, are noted.

Two of the organisms (*Bacillus aroideae* and *B. carotovorus*) that induce bacterial soft rot of potatoes also cause decay of the stems of growing potato plants, producing a soft disintegration of the tissues that may or may not be black.

Bacterial soft rot occurs in potatoes from all sections and probably causes more destruction than do all other potato parasites combined, because it may occur as either a primary or a secondary rot. As a primary disease it is found most abundantly in the early southern immature crop, particularly when badly bruised or skinned potatoes are loaded while hot or wet and shipped with poor ventilation. (Pl. 4, A, B.) As a secondary rot it may follow other diseases or injuries, especially late-blight tuber rot, blackleg, Fusarium tuber rot, leak, brown rot, scald, freezing, and mechanical injury. In the field it often becomes important in water-logged soils as seed-piece decay.

CAUSAL FACTORS

Generally the soft rots of potatoes found in nature are induced by mixtures of one or more of the organisms which can attack live potato tissues, or of one or more of these parasites associated with other bacteria which alone can not attack the live tissues. The presence of the latter tends to modify the symptoms, however. This probably accounts for the fact that the bacterial soft rots produced in the laboratory with pure cultures are practically odorless in contrast with those occurring naturally, which often are vilely odorous. This mixture of organisms may also account for the wide variations in consistency and color of the rots both in nature and in the laboratory. It has been demonstrated experimentally that at least three species or varieties of bacteria isolated from soft-rot lesions on potato tubers can cause rot in pure culture. These are *Bacillus carotovorus*, *B. aroideae*, and *B. mesentericus*. In addition, *B. phytophthorus*, the causal organism of blackleg, and *B. solanisaprus* often induce serious and abundant soft rot of tubers, which can not always be readily differentiated from the other bacterial soft rots. (Pl. 3,

[10] The rot induced by *Bacillus solanisaprus* Harrison (syn., *Erwinia solanisapra* (Harrison) Holland; see Blackleg, p. 11) often exudes a clear liquid. In black rot the early stages are white, but later, especially in light and air, the border turns pink, and then jet black.

A, D.) It has been demonstrated that in addition to potato tubers, *B. aroideae* and *B. carotovorus* attack a wide variety of truck crops—bean, cabbage, carrot, celery, cucumber, eggplant, muskmelon, onion, parsnip, pepper, radish, salsify, tomato, and turnip.

The soft-rot bacteria are normal inhabitants of the soil. Like other soil organisms, their relative abundance can be increased greatly by accumulation of vegetable litter. They are so abundant that there probably are no vegetable crops on which they are not present, owing to washing, splashing, and blowing of the soil. Furthermore, plant tissues are contaminated with these bacteria during harvesting and packing by means of soil, tools, hands, or wash water. Not only are they present as contaminants in the field, but they cause active rot there, as in cabbage, muskmelon, tomato, and in root crops. Much of the rot that develops in transit and in storage has its actual inception in the field.

Since all of these organisms attack potato-tuber tissues through breaks in the protective coverings, practically all invasions of tissue by the soft-rot organisms are through mechanical wounds, the lesions of other diseases, or tissues weakened by mechanical agencies, by freezing, or by aging. Soft rots are the most common invaders of aging or yellowing leafy tissues and of tissues affected with nonparasitic pathological conditions, such as freezing injury, sun scald, and black-heart, or with parasitic diseases, such as bacterial wilt or late blight.

The organisms will invade only moist and turgid tissues and can attack dead or injured tissues only when they are wet. Breaks in the tissues or moistened surfaces provide them with the water and food they need to get started and to carry on growth and reproduction. The rot may be spread in transit or storage by contact or by oozing and dripping from the decayed tissues. There probably is much less actual spread of this decay in transit than of many fungus rots, because there is no aerial mycelium. *Bacillus carotovorus* is particularly sensitive to desiccation and to direct sunlight.

Wet weather favors the disease in the field directly and indirectly. It brings about a multiplication of the organism, disseminates it, moistens plant tissues, makes the plant tissues more turgid, and often suffuses them with water so that they break or crack.

The temperature relations of *Bacillus aroideae*, *B. carotovorus*, and *B. mesentericus* are not identical; consequently, temperature to a great extent will determine which organism will invade a given lot of potatoes. *B. carotovorus* attacks potatoes at temperatures ranging from 46.5° to 92° F., but causes comparatively little rot outside the range 70° to 84°, most rot occurring between 70° and 77°; *B. aroideae* invades tubers beginning at 52° and is a very active producer of rot at temperatures between 68° and 102°; whereas *B. mesentericus* invades tubers only slightly at 53.5° but is active between 68° and 107.5°. At 98° *B. carotovorus* no longer induces rot, while *B. aroideae* and *B. mesentericus* induce very active rot. At 102° *B. aroideae* no longer causes rot, while *B. mesentericus* causes rot at the upper limit of temperatures tolerated by potato tissues. Consequently, *B. carotovorus* is a potential danger in cool storage, while the other organisms are most significant in tubers that become heated in the field, in transit, or in storage.

Bacillus phytophthorus, the causal agent of blackleg and of a soft tuber rot, invades tubers at temperatures between 35.5° and 92°, being most destructive between 42° and 84°. *B. solanisaprus* induces slight rot at 37.5° and active rot beginning at 68° and extending above 89.5°. Its lower and upper temperature limits have not been determined. At a temperature around 87° all the organisms cause active rot.

It has been experimentally demonstrated that in very dry air there is little infection by any of these organisms through either surface or deep wounds, and that in practically saturated air each of the organisms can infect through a surface wound or a deep wound at temperatures of 68° to 77°. Relative humidities below 80 per cent retard progress of the rot even after it has become established by way of deep wounds.

Striking differences in resistance and susceptibility to the rot have been established experimentally for tubers of different potato varieties. Ten varieties tested showed the following ranking on the basis of increasing resistance to bacterial soft rots: Rural New Yorker, Russet Rural, Burbank, Irish Cobbler, Green Mountain, Triumph, Early Ohio, Early Rose, McCormick, and Spaulding Rose. Burbank, Early Ohio, Spaulding Rose, and Green Mountain, after prolonged storage, are more subject to rot induced by the blackleg organism than to the soft rots induced by *Bacillus aroideae*, *B. carotovorus*, and *B. mesentericus*. Spaulding Rose is resistant to all rots save that induced by the blackleg organism.

CONTROL MEASURES

As in many other storage rots, the control of bacterial soft rots begins in the field. One step is the prevention of diseases of the growing or maturing crop, because these increase the reservoir of infection of the bacterial soft rots by providing dying or dead plant material. Furthermore, field diseases produce lesions in tubers through which soft-rot infection may occur, as in late blight, brown rot, or blackleg.

Crop rotation will not only aid in the control of certain field diseases, but will also lessen the reservoir of soft-rot bacteria in the soil. The reservoir of contamination in the field may also be greatly reduced by a reasonable degree of sanitation in the disposal of culls, trash, and litter. In packing, rotting tubers should be sorted out, and these and other rotting vegetables should not be thrown on manure or compost heaps to be eventually returned to the field. In fact, every precaution should be taken to guard against the use of contaminated manure or compost. Proper handling during harvest to avoid all unnecessary wounds, cuts, bruises, sun scald, and freezing injury is of primary importance in the prevention of soft rot. The potato, like many root crops, may profitably be allowed to dry before being stored or shipped. This will kill the soft-rot bacteria on the surface of the tubers and allow all surface wounds to become dry.

Ventilation and low temperature during transit and storage are, of course, most important factors in the control of this rot. Ventilation, by reducing the relative humidity and preventing condensation

of moisture on surfaces, renders the tissues less turgid and so less susceptible to infection. Furthermore, ventilation disposes of the products of respiration, including heat, and prevents black-heart, which predisposes potatoes to soft-rot infection. Storage pits, bins, and houses should be properly constructed so as to provide for the necessary ventilation. Where actual refrigeration is impossible, much can be accomplished by properly ventilated cool storage and transit conditions. Temperature control during storage and transit is probably relied upon to a greater extent than any other factor for the prevention of soft rot. Soft rot will not make any progress in refrigerator cars after the products have once become thoroughly cooled.

Because of the offensiveness of the bacterial soft rot, salvage and reconditioning of partially affected lots of potatoes is less feasible than it is with the fungous rots. However, by careful sorting apparently worthless shipments may be salvaged provided whatever wounded stock is obtained can be used at once or exposed to dry air, especially in direct sunlight. (*17*, *21*, *59*, *72*, *82*, *89*, *92*, *104*, *115*, *167*.)

BLACK-HEART

Black-heart is the result of abnormal conditions in and subsequent death of the tissues of the potato tuber, with definite post-mortem changes, induced by a deficiency of oxygen. It may be brought about at any temperature by an absolute lack of oxygen, as a result of inadequate aeration, or in full supply of air if the temperature is so high that it causes a rate of oxygen consumption by the tissues in excess of that supplied by normal air through the outer tuber tissues. These conditions result in death of the cells, the most secluded ones of the pith and phloem parenchyma dying first. Death of the cells is followed by discolorations, the ultimate jet black noted in affected tubers having given rise to the name of black-heart. The discolorations are the result of unregulated interaction among various constituents of the normal cell and free oxygen. The enzyme tyrosinase acting upon certain pigment-forming substances in the presence of free oxygen leads to the development of a group of substances, among them melanin, which give the pink, brown, and black colors characteristic of the various stages of black heart. (Pl. 5, C, D, E.) It should be borne in mind that the colors noted in black-heart can be found quite generally in killed potato tissues. There is some suggestion of them in freezing injury (see p. 17) and almost a duplication of them in leak (p. 33), pink rot (p. 35), and in some types of Fusarium tuber rot (p. 21). They are noted also in "scalded" tissues. (See Scald, p. 46.)

Black-heart is not known to occur in growing tubers, except occasionally in flooded soils. It occurs in the field, however, after digging, when tubers are allowed to lie in or on hot soil exposed to the sun. It occurs in transit when the temperature in stove-heated cars is allowed to go over 90° F. It occurs also in storage houses and pits if the piles of tubers are made so large that the interior can not be well aerated. Under those conditions it may even occur in refrigerated rooms, especially if the tubers are hot and wet when placed in storage.

A, B. Chemical injury

C, D, E. Black-heart. The changes in color from normal through pink and brown to jet black, characteristic of black-heart, often are found in potato tissues affected with freezing injury, scald, leak, pink rot, and Rhizopus rot

F. Greening, or sunburn, caused by exposure to light

G. Enlarged lenticels

The symptoms of black-heart vary, depending upon whether the tubers were exposed to high temperatures with a normal air supply, or to high, low, or normal temperatures with an insufficient air supply. In the former case no external symptoms develop; in the latter, both external and internal symptoms appear. The killed tissues are easily invaded by bacteria and fungi, which may obscure typical black-heart symptoms by causing various forms of watery or slimy decay. The external symptoms of black-heart are moist areas on the surface which may be purplish at first but turn brown or black within a short time. The internal symptoms are a dark-gray to purplish or inky-black discoloration. Injured tissues are of normal color when freshly cut soon after the injury of the tuber, but shortly after the air strikes them they turn pink, then gray or purplish, and finally jet black. (Pl. 5, C, D, E.) Sometimes all the colors except pink are found simultaneously in the same tuber; at other times only gray or brown are found, which is the case when tubers are heated above 130° F., or when they are deprived of oxygen for considerable periods after death, as in water-logged soils or in flooded pits. The colors usually are sharply set off from the healthy tissues. The affected tissues are firm and even leathery if they have dried a little—quite unlike those affected with leak, which frequently show colors similar to those of black-heart. When the discoloration is due primarily to insufficient oxygen it is restricted to the heart of the tuber, but it may radiate to the exterior as well. When one-sided exposure to high temperature is involved, as in a stove-heated car or in exposure to the sun in the field, the discoloration may affect only the exposed side of the tuber. (Pl. 5, D.) The discolored regions may appear in zones in the outer parts of the tuber and be absent or less evident in the center. In advanced stages the tissues dry out and cavities result. (Pl. 5, E.)

Since tubers will not usually develop black-heart at temperatures below 95° F. if given a good supply of air, control of the injury involves storage at temperatures below 95° with good ventilation. In stove-heated cars or storage places the tubers near the stove should be protected by tin sheeting. The temperature in heated cars should not be allowed to go above 60° or 70°. To prevent a shortage of oxygen, tubers should not be left long in hot, light soils after the vines are dead or be left lying near or on the surface after digging during hot weather. Flooding fields or packing hot, wet, or soil-covered tubers in closed containers, such as barrels, should be guarded against, since these methods may lead to asphyxiation. (*14*, *15*, *16*, *29*, *174*.)

BLACKLEG (TUBER ROT)

(*Bacillus phytophthorus* Appel)

OCCURRENCE, SYMPTOMS, AND EFFECTS

The disease known as blackleg of potatoes occurs in practically all parts of the United States and is most abundant in wet, warm seasons. It affects all parts of the plant and is of importance not only because it kills the growing plant and thus reduces the yield, but also because it affects the tubers, rotting them in the field, in transit, and in storage, and opening the way for other bacterial soft rots. (See Bacterial Soft Rots, p. 6.)

The disease derives its name from the fact that the lower portions of affected stems are characterized by relatively dry jet-black lesions. The tops of affected plants have an upright, staring appearance, due to a hardening of the vascular bundles, and generally are yellowish. If the plant is attacked early it generally is dwarfed and dies without any tuber production. If infection takes place later, a crop of tubers may be produced, some of which may become infected through the stolon from the mother plant. In irrigated sections infection often proceeds from the crown of the plant. In these cases the pith of the plant is destroyed, and dead black areas often run far up into the stems, extending to even the outermost leaves. Infection can also proceed through the lenticels, especially in wet soils.

When new tubers contract the disease from the mother plant through the stolons, the stem end of such tubers is the first to show symptoms. The early symptoms are browning and softening of the tissues about the vascular ring. In very moist places, especially under irrigation conditions in the West, tubers also become infected through the lenticels by contact with diseased ones. Some spread of the inoculum may take place in storage by this method. The decay often proceeds into the pith, destroying it completely and leaving a hollow with a black and slimy lining. (Pl. 3, A.) In the long western varieties the decay may develop as a stem-end rot, producing a shriveled, shrunken external appearance. (Pl. 3, D.) Often the entire tuber is converted into a pulp which in freshly decaying tissues may be white or only slightly colored, and cheesy or buttery in consistency. (Pl. 4, B.) Gradually this pulp becomes slimy and black as decomposition progresses. (Pl. 3, A. D.)

In typical tuber rot induced by *Bacillus phytophthorus* the decaying tissues at first are white or yellowish, but later turn pink and jet black along the border. Often there is a fishy odor. These characteristics generally serve to differentiate this rot from the bacterial soft rots. (See p. 6.) The decay induced by *Bacillus solanisaprus* (an organism closely related to *B. phytophthorus*) is soft and white at first, and either remains so or becomes slightly brown upon exposure to air. There is no border which turns from white to pink to jet black, and there is no disagreeable odor. These characteristics serve to differentiate this rot from typical blackleg rot due to *B. phytophthorus*. When *B. solanisaprus* attacks live stems it does not produce black lesions as does *B. phytophthorus*, but green-colored ones if it produces rot at all. The tissues decayed by *B. solanisaprus* often exude a clear liquid, and this characteristic may serve to distinguish it from the bacterial soft rots caused by *B. aroideae*, *B. carotovorus*, and *B. messentericus*. *Bacillus phytophthorus* and *B. solanisaprus* play an important rôle in tuber rots under cool-storage conditions.

In addition to the potato tuber and potato stems, *B. phytophthorus* rots the following vegetables: Bean, cabbage, carrot, tomato, and turnip.

CAUSAL FACTORS

It is well established that typical blackleg is induced by the bacterium *Bacillus phyiophthorus*, and it is fairly well established that

the closely related organism *B. solanisaprus* induces similar symptoms in potatoes. It has been shown that *B. phytophthorus* can overwinter in tubers in storage and that it is carried into the field by them. The inoculum is frequently spread in the preparation of tubers for planting by being carried over from infected to sound tubers. This occurs more commonly than is surmised, because infected tubers are not necessarily discolored or decayed. Whether the organism overwinters in the soil and is a factor in causing infection is a moot question. Observations and experiments in Minnesota and in western potato sections indicate that this is possible, while experiments in the East indicate that the organism does not overwinter in the soil, except in so far as it is associated with the seed-corn maggot. It is well known that the young sprouts become infected from diseased seed pieces, which invariably decay into foul-smelling masses of pulp. Since the organism can enter through lenticels, in wet soils the decay spreads from tuber to tuber by contact, and there is every indication that irrigation water carries the organism from hill to hill. An abundance of moisture is necessary for the development of the disease in the field. In addition, moderately high temperatures are essential.

The seed-corn maggot (*Hylemyia cilicrura* Rondani) and the potato-seed maggot (*H. trichodactyla*) play an important part in infection of cut seed pieces in the air and in the soil. If cut pieces are placed in excessively wet soil there is poor development or no development of wound cork, and the tissues are readily affected by rots of diverse causation. In the soil the cut surfaces and the decaying tissues of such seed pieces attract the yellowish-white legless maggots of these insects, if they are not already present, and they burrow over the surface and through the seed pieces. The surface of the eggs of these insects is often infested with the blackleg organism, which eventually gets into the intestinal tract of the larvæ and is thus carried into the tuber tissues by the maggots.

The rôle of temperatures and humidities in tuber decay induced by these organisms and experimental data in relation to the resistance and susceptibility of tubers of various potato varieties to *Bacillus phytophthorus* are discussed under Bacterial Soft Rots (p. 6).

CONTROL MEASURES

The only method of control in the field is to rogue all diseased plants as soon as they appear. In the eastern potato sections the most severe attack of the disease occurs in the early part of the growing season. In the irrigated sections of the West the new infections appear throughout the growing season, practically up to digging time. After the harvest all diseased tubers should be discarded, because they are likely to decay and spread the infection to their neighbors. Tubers from infested hills should not be used for seed even when they appear sound. Discolored or decayed tubers, of course, should not be used. In case one has cut into an infected tuber, the knife should be disinfected before further use. Treatment with either formaldehyde or corrosive sublimate solution is regarded as advisable to disinfect the surfaces of tubers that may have become contaminated with the organism through contact with

diseased tubers in handling or in storage. For control of the tuber rot in transit and storage, the discussion of bacterial soft rot (p. 6) should be consulted. (*7*, *18*, *19*, *21*, *70*, *79*, *84*, *85*, *86*, *113*, *115*, *129*, *130*, *142*, *150*, *166*, *171*.)

BROWN ROT (SOUTHERN BACTERIAL WILT)

(*Bacterium solanacearum* (E. F. Smith) E. F. Smith)

OCCURRENCE, SYMPTOMS, AND EFFECTS

In addition to the potato, all varieties of which are susceptible, brown rot or bacterial wilt occurs on eggplant, pepper, tomato, tobacco, jimson weed, black nightshade, peanut, and other plants. It apparently does not occur in the northern half of the United States. In the South it seems to be most serious in the first crops planted on virgin soil.

From the point of view of production, the losses are due to early death of plants, so that they produce either a diminished crop or none. The quality of fruit produced by sick plants is likely to be inferior, owing to underdevelopment. Losses are not restricted to the field, however. In the potato, affected tubers may merely be discolored and as a result bring a reduced price, or they may be invaded by secondary organisms and decay. (See Bacterial Soft Rots, p. 6.)

Brown rot or bacterial wilt occurs most frequently in the warmer sections of the United States and for this reason is often referred to as southern bacterial wilt. The parasite seems to be native to some of the virgin soils of the South Atlantic and Gulf States. Consequently the disease is important, especially in crops grown on new land in Alabama, Florida, Georgia, Louisiana, Mississippi, North Carolina, South Carolina, and Virginia. It is not limited to the United States, however, for reports of serious losses come from the Tropics.

Under transit conditions brown rot is a common and frequently a serious factor in shipments of the early or new crop from States on the Gulf and Atlantic seaboard. Since practically none of this crop goes into storage, the disease is of little importance as a storage rot. On the tuber the external symptoms of brown rot may appear as a slight depression at the point of attachment to the stolon, or as a grayish-brown discolored patch on the surface. (Pl. 4, D, E.) Sometimes no external symptoms develop. Upon cutting across infected tubers a moist, brown discoloration and a slight softening of the water-conducting tissues are seen. Frequently, though not invariably, yellowish-white, sticky globules of bacteria ooze out from the discolored tissues. (Pl. 4, C, E.) At this stage there is no odor. Later, as the bacteria work toward the center of the tuber, the entire interior becomes soft and brown, while a thin crust of tissues holds the tuber together. If the rot reaches this advanced stage while the tuber is still in the soil the shell is usually broken when the tubers are dug, and the bacteria-filled contents are left in the soil. If the rot reaches this stage during the period of transit or while the potatoes are in storage, neighboring tubers are likely to become smeared with the rotten contents. In the later stages of the disease other organisms gain entrance (pl. 4, D), the tissues are converted into a creamy consistency, and an indescribably foul odor results.

In the growing plant the outstanding symptom is wilting, which appears rather suddenly. At first it may be restricted to a single shoot, but generally it ultimately affects the entire plant. Sometimes the foliage yellows before it wilts, but if the onset of the disease is sudden, wilting occurs without previous yellowing. The stems shrivel if they are not very woody, usually turning yellowish green at first and finally becoming brown or black. A cross section of the stem or leaf petiole shows brown dots or a brown ring, and the discolored vascular tissues often show through the green parts of the stem and petioles as long, dark streaks. Closer examination shows that the bundles are filled with bacteria, which may ooze out upon cutting. The presence of the bacteria in the water ducts causes clogging, and if the bacteria occur suddenly in great numbers the plant wilts quickly without becoming yellow.

CAUSAL FACTORS

The bacterium that induces brown rot (*Bacterium solanacearum*) seems to occur in certain virgin soils of the South. This statement is based on observations to the effect that the disease occurs in potato crops and other solanaceous crops grown from disease-free seed, on land that had never before produced potatoes or even members of the family. In fact the disease is most severe in potato crops grown on new soil and diminishes in frequency and severity the longer the soils are cultivated and cropped. The bacteria in the soil attack the roots and underground stems of plants and thus gain entrance to the conducting tissues, along which they travel or are carried to all parts of the plant. If the soil is free from the organism, the latter may be carried into a field by insects from diseased crops in near-by fields. Once a single plant becomes diseased in a field, neighboring plants are in danger, unless leaf-cutting insects are held in check. It has been reported that when the Colorado beetle was allowed to feed on diseased plants and was then removed to healthy plants to feed on the tops, symptoms of the disease appeared in these plants in seven to nine days. It has not been demonstrated whether other insects, such as flea beetles or blister beetles, may spread the disease. Neither is it known whether the organism can enter through the unbroken surface of the plant.

Tubers get the disease from the infected plant through the water-conducting tissues. In a greenhouse experiment, three weeks after symptoms appeared in the tops the tubers were found to be entirely or partly rotted. There is no spread of brown rot from tuber to tuber in the marketing process, but it is probably true that the bacterial rot which follows does spread by contact. (See Bacterial Soft Rots, p. 6.)

Warm, moist weather favors the development and spread of the disease. The parasite grows well at temperatures ranging from 77° to 97° F. As the temperature is reduced the rate of growth is retarded, and growth is practically stopped at 55°. The disease consequently can not develop at temperatures below 55°, and it develops most rapidly between 77° and 97°. It seems to progress most rapidly in young plants. Unless infected tubers are held below 55° in transit, they are likely to decay.

CONTROL MEASURES

Satisfactory control measures can hardly be recommended until more is known about this disease. The most serious cases apparently occur on virgin soils, and when planting under these conditions growers should realize the risk. The question as to whether or not continuous potato cropping on the same fields tends to increase the chances of disease needs investigation. Some Florida growers have reported that with continuous cropping the danger of brown rot diminishes.

Tubers that are known to be diseased should not be stored or shipped, even though the trouble may be only in the initial stages, for such potatoes rot very quickly at temperatures above 55° F. As it is practically impossible to discover and remove all tubers that are in the early stages of the disease, potatoes from affected fields should be used immediately or shipped to near-by consuming centers, or else stored under cool, dry conditions, with good ventilation. (*105*, *169*, *170*, *173*.)

CHEMICAL (SALT) INJURY

Potatoes suffer from an injury caused by common salt, and some other chemical salts, such as fertilizers, produce the same effect. Injury is also caused by asphalt, coal tar, and oil products. In all of these mere contact with the chemical apparently is not sufficient to induce the injury. If the substances are ground into the tissues of the tuber by pressure or rubbing, however, the characteristic symptoms are developed. This occurs most commonly when potatoes in storage or in transit are piled on floors or in contact with walls that are impregnated with the chemicals enumerated. Affected tubers are unmarketable, and through the decay that often sets in they become a menace to sound tubers.

Usually the side of the potato in contact with the chemical is flattened (pl. 5, A, B), and the injured tissues are soft and flabby and at times semiwatery. Sometimes the chemical can be tasted within the killed tissues. The injured tissues frequently are firm and leathery, especially if they are not invaded by microorganisms. When the injury is first done the tissues are not discolored, but after a short exposure to the air they become brown to black and dry out. (Pl. 5, A, B.) The exterior of the killed area is usually black.

Control of this injury consists in keeping potato tubers from coming into contact with the chemical.[11] (*44*, *167*.)

DROUGHT AND HEAT NECROSIS

As the name implies, the pathological condition designated as drought and heat necrosis is considered to be due to drought or heat or a combination of both. Little more can be said about the causal complex until experimental work is done. The condition affects the tubers and lowers their market value.

This pathological condition has been noted in tubers grown in light, hot soils in the hot potato-growing sections of the United

[11] LUSK, J. P. INVESTIGATIONS ON SALT INJURY OF POTATOES. 1926. [Master's thesis. University of Chicago, Department of Botany.]

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A. Freezing injury, general type
B. Freezing necrosis
C. Internal brown spot
D. Heat and drought necrosis
E. Net necrosis

States. It has been noted especially in the early crop grown in the volcanic ash soils of Idaho, and it seems to occur in tubers that are allowed to lie in hot soils when the vines suffer from lack of water and begin to die. It is marked by the death and a golden yellow to brown discoloration of the vascular tissues and of the parenchyma immediately surrounding them. (Pl. 6, D.) The discoloration is most pronounced in the ring tissues, at either the stem or the bud end, and in the outer phloem tissues. It thus differs from net necrosis (pl. 6, E), which it somewhat resembles both in color and in distribution of the necrotic tissues. Discoloration at first is restricted to the vascular and immediately adjoining elements, but after a time it spreads slightly to surrounding parenchyma, and the color changes from golden yellow to a light or dark brown. Sometimes in light-skinned varieties the discolored tissues give a darkened appearance to restricted areas of the surface. Upon cutting, it is found that the discolorations are not due to a solid mass of tissue as they appear to be, but rather to the discolored strands, which impart a dark hue to the tissues overlying them. (Pl. 6, D.)

Control of the disease involves keeping the soil moist, cool, and shaded and in digging the tubers as soon as the vines begin to die, if the soil is light and the weather hot. (*167.*)

ENLARGED LENTICELS

Enlarged lenticels affect the marketability of potatoes, but they do not predispose them to decay, nor do they impair their food value. Under normal soil and atmospheric conditions the lenticel cells are dormant. In very wet or water-logged soil and in very humid atmosphere these cells enlarge and divide. If this growth continues long enough, a mass of cells is formed which protrudes above the tuber surfaces. (Pl. 5, G.) At first these cells are quite soft, whitish in color, and easily rubbed off; later they harden and become brown and corky. (*167, p. 22.*)

FREEZING INJURY

OCCURRENCE, SYMPTOMS, AND EFFECTS

Upon thawing, tubers frozen to death may show freezing injury so plainly externally that they are unsalable and unfit for table purposes. On the other hand, the injury from less severe freezing may be so slight that it is not apparent externally and is revealed only when the potato is cut, perhaps several hours after the exposure to low temperatures. This condition is commonly referred to as frost necrosis. Even though the nonaffected areas, which may be considerable, are fit for food, tubers so affected are not desirable for table purposes, because they are unsightly when served, or because the removal of discolored areas involves an inordinate waste of time.

Storage or shipment of frozen tubers or tubers affected with freezing injury involves hazards of total loss through secondary decays or at least through excessive wrinkling and shriveling due to drying out. The use of affected stock for seed purposes may lead to weak plants or to poor stands because of seed-piece decay.

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The symptoms of freezing injury vary, depending upon what kind of tissues are killed, the amount of tissues involved, and the consistency and color of the affected regions. Which of the symptom complexes will develop depends upon whether the injury is due to (1) long exposure to temperatures just below the freezing point, or to moderately long exposures to very low temperatures, or whether it is due to (2) a moderately short exposure to temperatures just at the freezing point or short exposures to temperatures far below the freezing point. In both of the former cases most or often all of the tissues may be killed, and both external and internal symptoms may appear shortly after thawing. In the latter case only the most susceptible tissues are killed, and no external symptoms are apparent for weeks or months. Short exposures generally produce slight injury of the net or ring types, or a combination of these; longer exposures the blotch type, often in combination with the net and ring types. The restricted or limited type of injury is known as freezing or frost necrosis.

Frozen potato tissue, before thawing, no longer possesses the crispness of the normal tissue. It is abnormally firm, looks dull, and does not cut readily or with snap. This is due to the presence of ice. When ice occurs in only part of the tuber, its presence at times can be detected by a crunching sound if, while held to the ear, the tuber is pressed between the fingers.

Tubers that are frozen solid collapse promptly upon thawing, becoming soft and watery. (Pl. 6, A.) Their wet surfaces make detection and sorting out easy. The presence of moisture on a tuber is not necessarily a sign of freezing injury, however, since cold tubers when brought into moist air with a temperature higher than their own very readily condense water from the air on their surfaces. Sometimes if only a portion of a tuber is exposed to freezing temperature the exposed portion is frozen to death and upon thawing becomes turgid and blisterlike, and the skin frequently discolors. If tissues that have been frozen to death are cut shortly after thawing, they may appear dull and colorless at first. Upon exposure to the air they frequently pass promptly through pink and red color stages and ultimately become brown, gray, or an inky black. (See Blackheart, p. 10.) The rate of development of these colors depends upon the temperature of the air. At temperatures below 75° F. several hours may be required, while only one-half to one hour may suffice at higher temperatures. The occurrence of these color changes is also determined by the extent to which air reaches the affected tissues.

When extensive areas of tissues are killed by freezing, they usually become infected with bacteria, which cause foul-smelling, slimy, or sticky rots if thawing takes place in a warm, humid atmosphere. They dry down to a mealy or tough, leathery, granular, chalky mass of starch and tissue remnants if they thaw in cold or dry air. Tubers that have only one side frozen frequently have the killed portion sharply set off from the nonaffected area by a purplish or ultimately brown line of corky tissues. (Pl. 6, A.) Often infection by Fusarium species sets in before the unaffected cells are sealed off by the corky layer.

It is generally known from experience with potatoes in the field, in storage, and in transit, and from experiments, that when a lot of

tubers has been exposed to low temperatures for a long enough time to freeze some solid, there frequently are others that do not freeze solid and subsequently collapse upon thawing, but which seem to be unaffected so far as external appearances indicate. If such tubers are cut they may show the various types of symptoms known as freezing or frost necrosis. (Pl. 6, B.)

The mildest type of freezing necrosis is one marked by tissues that are drier than surrounding tissues and that have a grayish-white tint. When the injured tissues are first cut soon after thawing, they may turn red in a short time and finally become brown. In uncut tubers, even after a long time, the only sign of injury is a grayish, dull, dry, collapsed appearance of some of the tissues.

Generally, however, freezing necrosis is marked by decided discolorations. These may not be apparent immediately after thawing, but they usually set in after five or six hours. The color changes usually from dull white to pink or brick red and finally to gray, brown, or black.

There are several types of discoloration. One is the ring type (pl. 6, B), which is limited to the vascular ring and immediately adjoining tissues and is characterized by pronounced blackening. Another is the net type, which is marked by more or less blackening of the vascular ring and the finer strands which extend from it into the inner phloem parenchyma and outer phloem parenchyma. (Pl. 6, B.) Both types are frequently restricted to the stem end. Finally, there is a blotch type, marked by irregular patches ranging in color from an opaque gray or blue to sooty black. These patches occur everywhere in the tuber, though most generally in the vascular ring and cortex. When these blotches are in the cortex they may be apparent externally in clean tubers with white skins. This is the only type of freezing necrosis that may be visible externally. If tubers affected with any or all of these types of freezing necrosis are held in storage, they generally shrivel or wilt more than nonaffected tubers. Excessive shriveling alone, however, can not be relied upon as a sign of freezing necrosis.

It is popularly believed that the symptoms here described as freezing necrosis are not due to freezing but to chilling, since freezing, it is contended, is always followed by complete breakdown after thawing. This is not the case. Freezing necrosis is as truly a freezing injury as the complete type and does not occur unless ice is formed in the tissues. The difference lies in the fact that not all cells of the tuber, but only those that become discolored, have been killed.

If tubers are exposed to low temperatures, but not low enough to cause ice formation, sugars accumulate, and the tubers become sweet. This sweetness disappears if the tubers are kept at temperatures above 40° F. Frozen tissues are no sweeter than uninjured ones. Sweetness of tubers, therefore, is not a sign of freezing, as is popularly believed.

Experiments show that the various tissues of the potato have a varying susceptibility to freezing. The vascular tissues are most easily killed, and therefore, with a given temperature and exposure, they frequently are injured, while the other tissues either do not form ice or thaw without visible injury. The vascular tissues of the stem end of the tuber are also more susceptible than those of the bud end.

Consequently, freezing necrosis usually proceeds from the vascular tissues and begins at the stem end.

Potato tubers will not freeze at 32° F., the freezing point of pure water, because the sap or juice of potatoes is not pure water but a solution of salts, sugars, and other more complex materials. The expressed sap freezes at about 30.8°. The freezing points of the tissue is even lower. This is probably due to the influence of the living cells and the capillary action of the intercellular spaces.

The average freezing point of 18 varieties of potatoes has been found to be 28.9° F. Varieties belonging to the early or midseason groups have a higher freezing point than those of the late-season groups, and it has been observed that the freezing point of a given variety falls as the season progresses. The freezing point also varies somewhat with the temperature at which the stock has been held. Generally speaking, a lot of potatoes stored at 32° for several weeks will stand about 1 degree lower temperature without freezing than a similar lot that has been stored at 50°.

It is impossible to forecast the critical temperature for an individual tuber and the time of exposure to low temperatures requisite for injury, because there is great variation in the individual susceptibility of tubers to freezing. Even though the critical low temperature—that is, the freezing temperature—for living potato tissue lies between 28° and 30° F., the temperature of tubers can fall below this point without ice formation, owing to the phenomenon known as undercooling. Associated with this is the phenomenon termed "inoculation," which consists of an instantaneous formation of ice and a sudden rise of temperature in the undercooled tissues to their true freezing point, following a mechanical disturbance such as jarring or a sharp blow.

The degree to which a tuber tissue will undercool depends among other factors upon the air temperature, the time during which it is exposed to low temperatures, and the rate at which undercooling takes place. The faster the temperature falls the less undercooling is tolerated by the tissues. Tubers exposed to air whose temperature dropped slowly to 23° F. undercooled to 23.6° and 23.1° after 165 and 174 minutes, respectively, before freezing, while others exposed at once to the same temperature undercooled only to 24.5° in 73 minutes. Tubers exposed to a temperature of 8.7° froze in 6 minutes, their temperature falling to 23°, while others, exposed to 15.8°, froze in 12.5 minutes.

Tubers whose surfaces are wet or which have newly made wet wounds frequently undercool only slightly if at all. That mechanical disturbance of undercooled tubers leads to freezing injury is proved by the following experiments: Tubers kept for varying lengths of time at 26°, 27°, and 28° F., respectively, were dropped through distances varying from 1 to 4 feet. They showed freezing necrosis. Others treated identically but dropped only 2 to 6 inches showed no injury. However, when struck with a pencil they froze. Continuous bouncing through a distance of 1 inch for 10 seconds produced similar results. Tubers kept at 28° were rolled 30 and 50 feet in a bag. Many of them showed injury. In all cases the controls—that is, tubers kept at the same temperature but not subjected to dropping, striking, or rolling—remained uninjured. In this experiment varietal and individual susceptibility again were apparent.

Frozen tubers should be sorted out as much as possible, since they are subject to severe drying and shriveling as well as to secondary rots, which may become a menace to the unaffected tubers. It is, of course, impossible to sort out tubers that do not show any external symptoms. In case there is reason to suspect freezing injury in a lot of potatoes it is advisable to cut some tubers to determine the presence or the extent of the trouble.

If an affected lot is sold, either a rejection of the entire lot or a demand for an allowance may be expected, since affected stock is not desirable for table, seed, or storage purposes. Sometimes an affected lot reaches the consumer before any difficulty is experienced, and at times it may even go into consumption without complaint. Affected tubers can be used for stock feed and the preparation of potato starch.

CONTROL MEASURES

The prevention of freezing injury involves the protection of tubers against exposure to temperatures lower than 30° F.

Tubers should be dug before severe freezing temperatures set in. If they can not be removed to shelter at the onset of freezing weather, or if they are loaded or unloaded during cold weather, they should be protected by straw, canvas, sacks, and the like. If they are pitted, the covering should be sufficient to prevent internal temperatures below 30° F.

In cars or storage places when the temperature is likely to fall below 30° F. artificial heating must be resorted to. When this is the case it is essential that the potatoes be set away from the walls and above the floor by a false floor, so as to permit ready movement of air. (*76*, *78*, *98*, *193*, *195*, *196*.)

FUSARIUM TUBER ROTS

(*Fusarium* spp., primarily *F. trichothecioides* Wr., *F. eumartii* Carpenter, *F. radicicola* Wollenw., *F. oxysporum* Schl., *F. sulphureum* Schl., and *F. coeruleum* (Lib.) Sacc.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Although Fusarium rots occur in a wide range of truck-crop plants and are an important factor, especially in storage and during marketing, serious study has been made only of those that affect the potato and the sweetpotato. Generally Fusarium rots are most common in the tubers. When these fungi attack other parts of growing plants the symptoms complexes have received other descriptive names such as foot or root rot, wilt, or yellows. In most cases the parasite has not even been isolated or identified, and naturally little exact knowledge is available concerning the relation of environmental factors to the rots. This discussion of the rots of the potato, while not strictly applicable to those of other hosts, covers the general principles.

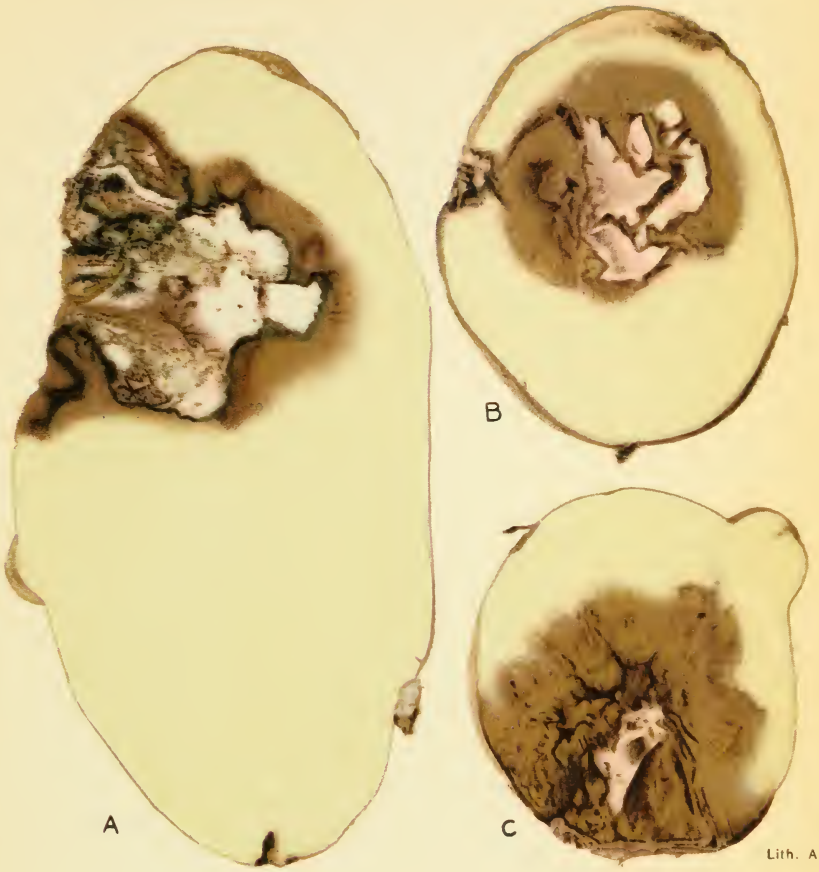
Unlike the Fusarium wilts, Fusarium tuber rots of the potato occur in potatoes from all parts of the United States. With the exception of stem-end rot (*F. eumartii*) and black field rot (*F. radicicola*), they do not occur in the growing tuber in the field. They are destructive in tubers primarily during storage and transit,

and the losses caused by them probably are exceeded only by those due to bacterial soft rot.

Some of the species that cause tuber rot also attack the growing plant, destroying the root parenchyma or invading the vascular elements or both, and cause blight or wilting. (See Fusarium Wilts, p. 25.)

Although the symptoms depend in part upon the species of the parasite and upon external conditions, especially temperature and moisture, a fairly general characterization of the rots can be given. The disease generally is marked by sunken, shriveled, wrinkled, or broken areas occurring on the tuber surface at the stem end (*Fusarium radicicola*, *F. eumartii*, and *F. trichothecioides*), at the eyes, at the lenticels, or anywhere between them. (Pl. 8, A, B, D; pl. 9, A.) As a rule these areas are brown to black, and on them may appear masses of whitish, dark, or brightly colored mold. The tissue underlying such discolored and sunken areas varies in discoloration and consistency, depending upon the species causing the rot and upon the conditions under which the rot develops. The rot may be wet and jellylike (jelly end rot, *F. radicicola*, pl. 9, C), even mushy and leaky (*F. oxysporum*, pl. 7; *F. radicicola*, pl. 8, A), or dry and brittle, and with or without cavities lined with whitish or brightly colored, fluffy or powdery molds (pl. 8, C). The color of the affected tissues varies from light to dark brown and even black. Sometimes several species attack the same tuber, causing a combination of symptoms. Fusarium tuber rot is usually dry at low temperatures and wet at high. It never is slimy, even when wet, and never has a bad odor unless accompanied by other fungi or bacteria. In some types of the rot, as in powdery dry rot (pl. 8, C) for example, the fungi have a tendency to rot the pith and inner phloem parenchyma faster than the tissues outside the vascular ring, so that a spot which is scarcely apparent on the surface may be underlain by an extensive wet region or by a rotted, dry interior, full of cavities and surrounded by a shell of healthy cortical tissues. (Pl. 7, B.) In other types the rot progresses more evenly into the tuber, destroying the various tissues at more or less equal rates. In some types the dried-out stage is often marked by layerlike, dry, tough remnants of tissues. In some types there is merely vascular discoloration (*Fusarium oxysporum*, pl. 9, D; *F. eumartii*); in others there is discoloration of the vascular tissues in advance of the rot, appearing as a network or flecks; and finally there is stem-end rot with vascular discoloration, accompanied by a glassy appearing brown rot of the adjacent tissues, which often extends throughout the length of the tuber (*F. eumartii*, pl. 9, E).

Unlike many diseases that are caused by generalized parasites and do not have lesions with diagnostic characters, but in which the parasites themselves are characteristic signs, the various Fusarium rots of potatoes and other crops can not always be identified by the structures of the parasite. Superficial mycelium does not occur invariably; when it does, it may occur as a cottony mass, which may remain white or may take on distinctive bright colors, or it may break through the surface in little tufts of white or brightly colored molds (pl. 8, A, B, D), as in some potato and onion rots. Neither the mycelium nor the spores are ever black.



A, B, C. Fusarium tuber rot, wet type (Fusarium oxysporum and Fusarium trichothecioides)

Lith. A. Hoen & C

A. Fusarium tuber rot (Fusarium radicicola)

B, C, D. Fusarium tuber rot (Fusarium trichothecioides)

Lith. A. Hoen & Co

A, B. Fusarium stem-end rot (Fusarium radicicola)

C. Jelly end rot (Rhizoctonia solani, Fusarium radicicola, and other species)

D, E. Fusarium wilt (Fusarium oxysporum, Fusarium eumartii)

The tuber symptoms of Verticillium wilt are often indistinguishable from D

Their bright color is the most distinguishing macroscopic character, white, orange, and ocher (pl. 8, A, B, C) being some of the colors found in conidia, while the mycelium may be carmine, blue, or brown. Not all Fusaria produce sclerotia. These are rarely produced on host tissues, and when they occur they are not distinctive. There are no macroscopic reproductive structures like the sporangia in Rhizopus. In fact, one must resort to the microscope to make a diagnosis even when a brightly colored mycelium is involved. This does not always decide the matter, for the characteristic sickle-shaped, dorsiventral, septate conidia which frequently give color to the mycelium often are absent. Microconidia may occur but usually are not characteristic enough for positive identification.

CAUSAL FACTORS

Like other Fusarium species, those parasitic on potato tubers are inhabitants of the soil, where they live saprophytically on plant débris. Although it appears that no soils are entirely free from pathogenic Fusaria, various soils and localities seem to be characterized by either the presence or the predominance of certain Fusaria. In general, the Fusaria that induce wilts have high temperature optima. Consequently, the Fusarium wilts are limited to or predominate in sections or seasons marked by hot weather.

In the rots the type varies with different sections, being determined by the species of Fusarium that either characterize the particular section or happen to predominate during the growing season. Thus the Fusarium rot that usually occurs in the Eastern States is induced by *F. coeruleum;* the rot in the Southern States is induced by *F. oxysporum* and by *F. radicicola* (pl. 8, A; pl. 9, A, C), which also induces black field rot in Idaho and contributes to jelly end rot of the Burbank variety in the irrigated sections of the far West. (Pl. 9, C.) The powdery dry rot in the middle western plains and Mountain States east of the Cascade Mountains is induced by *F. trichothecioides*, which also occasionally induces wilt (pl. 8, C, D), while the rot in potatoes west of the Cascades is induced by *F. coeruleum.*

As pointed out before, four species are known to invade the tuber or plant principally in the field. *Fusarium radicicola* apparently can invade tubers through the lenticels. *F. oxysporum*, *F. eumartii*, and to a minor degree *F. trichothecioides* can invade the growing plant and from it the young tubers.

All the other parasites are wound parasites and consequently become important after harvesting. The rough handling of tubers after harvest naturally provides many points of entry. Infection proceeds from the mycelium by contact or from spores carried on the tubers, in the soil, on containers and implements, and in storage houses.

Whether or not infection will take place and the length of the incubation period are determined primarily by moisture and temperature conditions. The rate of formation of wound periderm and of suberization is most important in determining infection. At a storage temperature of 44° F. these form so slowly that they offer very little resistance to the growth of the Fusaria. In the Rural New Yorker variety it requires 3 or 4 days at 70°, or more than 10

days at temperatures below 50°, for the formation of wound periderm that is effective in excluding *F. coeruleum* and *F. sulphureum*. The various species of Fusarium have different temperature requirements. *F. oxysporum* and *F. eumartii*, which can also induce wilt, do not grow at all at temperatures below 50°, grow best at 86° and 78°, and cease growth at 100° and 95°, respectively. *F. radicicola*, which induces black field rot and also jelly end rot, does not grow at temperatures below 53°, grows best at 86°, and ceases growth at 100°. *F. trichothecioides*, which induces dry rot and some wilt in the plains and Western States, does not grow at temperatures below 41° or above 86°, and grows best at 78°, at which temperature it also induces most rapid rot. *F. sulphureum* and *F. coeruleum* are able to infect tubers and cause a progressive decay at the lowest temperatures feasible for storing potatoes.

Under most favorable conditions the incubation period is 4 to 5 days. In 10 to 15 days as much as one-half to three-fourths of medium-sized tubers may be destroyed. At 77° F. tubers frequently are completely destroyed in two to seven weeks. At low temperatures (50° or below) most Fusaria grow only slowly, but the growth made at these temperatures may be sufficient to start infection and to establish the organism so that it is ready to make very rapid growth as soon as it is exposed to higher temperatures. Most of the rot-producing Fusaria that have been studied can endure temperatures below 41° or even freezing and remain viable for long periods.

CONTROL MEASURES

Control of the Fusarium diseases is not an easy matter. In the first place, the wilt diseases and the decays require different treatments. Rotation of crops has proved beneficial with some of the wilts, but in general it has not been as helpful as in other diseases because of the saprophytic habit of the organisms and their ability to feed on a wide range of hosts. The selection and development by breeding of resistant strains or varieties has proved a much more promising method of control with a number of other crops.

Although all soils, even virgin soils, seem to be infested with several species of Fusarium, it is well known that pathogenic species have either been introduced or made economic factors by cultural practices. Failure to rotate crops frequently makes the Fusarium problem acute. The light soils of the South that are suitable for watermelon culture usually will produce only one crop of watermelons free from wilt. It is well known that the powdery dry rot of potatoes is a far more serious storage rot in cellars in which rotting tubers were stored or allowed to accumulate than in new ones or in those which are kept clean and free of débris and in which a gaseous or liquid disinfectant is used.

The safest and surest methods of controlling the rots that are induced by wound parasites are the careful handling of tubers to avoid cutting and bruising, the sorting out of bruised, broken, cut, diseased, or frozen potatoes, and storing the tubers in well-ventilated places at temperatures between 36° and 40° F. Wound infection and rot can not be prevented in storage potatoes by manipulation of moisture and temperature conditions alone. (*25*, *49*, *87*, *90*, *111*, *137*, *168*, *186*, *190*, *191*.)

FUSARIUM WILTS

(*Fusarium oxysporum* Schl.; *F. eumartii* Carpenter; *F. trichothecioides* Wr.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Fusarium wilts occur in the warmer potato sections of the country or during warm seasons. Fusarium wilts of the potato not only reduce the yield by killing plants prematurely but also cause discoloration and decay so that tubers become unmarketable. In addition they open the way for aggressive tuber-destroying fungi. Discoloration in the vascular bundles without decay counts heavily against seed stock.

In other plants, Fusarium wilts generally are important because affected plants, if not killed outright, are discolored, or when they are free from lesions, are small, flabby, or ill-shaped, and thus have a lowered market value.

In the tubers the disease is marked by mere brown discoloration of the vascular ring (pl. 9, D, E), or it may be a stem-end rot of the tuber (pl. 9, A. C, E). In the case of stem-end rot (*Fusarium eumartii*) the sunken areas at the stem end occur with wrinkling around the margins. (Pl. 9, A.) When there is only discoloration, cutting is necessary to make it visible. In the field as much as a quarter of the tuber may became involved from the stem end. The rot displays a variety of symptoms: (1) Stem-end rot with no vascular discoloration and a sharp line of demarcation between the healthy and infected tissues (pl. 9, A, B); and (2) stem-end rot with vascular discoloration which may be associated with a soft rot of the adjacent tissues and may extend the entire length of the tuber as a band of light-brown tissue. (Pl. 9, D, E.) (See Fusarium Tuber Rots, p. 21.)

In the growing plant *Fusarium oxysporum* and *F. eumartii* induce somewhat different symptoms. The wilt induced by *F. oxysporum* is marked by yellowing around the margins of the lower leaves, which gradually develops in the entire top, accompanied by complete wilting. This yellowing and wilting usually affects one side of the plant more severely than the other. In the wilt produced by *F. eumartii* the first symptoms are light-green mottling of the leaves accompanied by the irregular bronzing of the upper surface. Under dry conditions the leaves slowly turn yellow; in severe cases there is leaf dropping, with the leaves hanging to the stem by a mere thread. Sometimes an internal blackening of the veins, which may be visible through the tissues, develops in the leaf blade and petiole. Under dry conditions the wilt progresses rapidly, and the entire plant wilts and dies in a short time. If there is an abundance of moisture, wilting does not result, but there is a yellowing, rolling, and rosetting of the top, which may be accompanied by the development of aerial tubers. In both types the fine roots are destroyed. Under wet conditions the cortex sloughs off, and under dry conditions the roots become brittle. In the former case there is discoloration of the vascular tissues of the stem, which, however, does not extend above the ground. In the latter there is brown discoloration of the vascular bundles, which is accompanied by necrosis of the inner and outer phloëms, and the discoloration ex-

tends into the upper stem and foliage, often being very pronounced in the pith at the nodes.

Frequently, wilts induced by Fusaria can be distinguished from those induced by bacteria by the absence of bacterial ooze in the affected tissues, but very often they can not be distinguished save by cultural methods. Verticillium wilt of the potato and other plants can generally be distinguished from Fusarium wilt only by laboratory determination of the parasites. Verticillium wilt of the potato occurs north of the range of Fusarium wilt. (See Verticillium Wilt, p. 50.)

CAUSAL FACTORS

Potato wilt is generally induced by two species of Fusarium, *F. oxysporum* and *F. eumartii*, and occasionally by *F. trichothecioides*, which, like other Fusarium species, are inhabitants of the soil, where they live saprophytically on plant débris and from which they may attack the plant. In the potato the parasites may attack the plant either from the soil where they overwinter or from infested seed pieces. In the case of *F. oxysporum*, most of the infection apparently proceeds from the soil into the roots. In the other types wilt is reproduced much more frequently in plants arising from infected seed. Nevertheless, infection from the soil through the roots is generally more severe and consistent in the latter type. In both cases the fungus grows from the mother plant through the stolons into the new tubers. There is no spread of the disease in transit or storage from tuber to tuber. The killed tissues, however, are subject to invasion by secondary organisms.

The temperature requirements of *Fusarium oxysporum* and *F. eumartii* are somewhat different. The former requires at least 57° F., with heavy soil inoculation, to produce the disease. The most rapid development of the wilt takes place at temperatures above 86° and up to the growth limit of the fungus, which is about 95°. Infection is favored by high soil moisture, but after infection has set in development of the wilt is faster in dry soil. The lowest soil temperature at which *F. eumartii* can produce wilt is 52°, and the highest is 86°. The most favorable temperature for infection and development of the rot is from 68° to 77°. Infection occurs in wet and dry soils, taking place more readily in wet soil, but the wilt subsequently develops more rapidly in dry soil. Tuber infection takes place more rapidly in wet soil.

The symptoms of wilts in other plants are essentially those found in the potato wilts. In these, as in the potato, blighting or wilting, often accompanied by prompt or delayed death, may be the result of destruction of the roots or foot of the plant, of general or local plugging of the water vessels (tracheomycosis), or the killing effect of the toxic substances liberated by the parasite or by the interaction of host and parasite, or a combination of these. Some of the wilt producers are also parenchyma destroyers or rot producers.

Wilt may appear at any time in the season, and once it has appeared, death of the foliage may occur slowly and fitfully or very rapidly and suddenly. The disease may appear so early, especially when it proceeds from potato seed pieces or sweetpotato roots, that the sprouts are killed before they emerge from the ground. Fre-

quently it appears in young plants, which may die prematurely or live to maturity. Finally, it may not appear until a short time before the crop is made.

CONTROL MEASURES

Since infection may proceed from the soil or from infected seed potatoes, two lines of control must be followed for potato wilt. If it is known that a given tract of land has produced a wilt-affected crop of potatoes, a long rotation should be used before potatoes are planted there again. There is some possibility that virgin lands may be infested with the organisms. Fields on which crop rotation has been practiced should be used as far as possible. Infected seed should be discarded. Since the parasite in a tuber is not necessarily visible, and since not all tubers with vascular discoloration are infested with Fusaria, the surest way to get clean seed is to practice roguing in the seed plot or to purchase only certified seed. Since tubers get the disease from the mother plant, it frequently is possible to prevent invasion of the tubers by the parasite, by digging a week or two early if the disease has not yet extended into them. (*33*, *34*, *51*, *62*, *87*, *97*, *99*.)

(For additional control measures, see Fusarium Tuber Rots, p. 21.)

GREENING (SUNBURN)

Greening of the tissues of the potato tuber results from exposure to light, either in the field or after harvesting. It is more commonly found in immature than in fully matured tubers. Affected tubers are discriminated against in the market, because they are bitter and unfit for food.

Ordinarily, the green color is restricted to the layers of cells immediately below the skin. If exposure to the light is prolonged, however, chlorophyll develops in the deeper tissues. (Pl. 5, F.) Once the bitter taste accompanying the green color has developed, it does not disappear completely even though the greened tubers are removed from the light. Often the affected tissues become tough, leathery, and excessively yellow, without developing chlorophyll. (*176*, *p. 32*.)

GROWTH CRACKS, KNOBBINESS, AND SECOND GROWTH

It is generally assumed that deep cracks (pl. 10, B) on the surface of tubers, knobs (pl. 10, D), pointed ends (pl. 3, D), and second growth are the result of uneven or unfavorable growing conditions. Although it is true that these malformations may appear on all varieties in all sections of the country under unfavorable conditions of growth, it is also a general observation that these deformities are produced under identical soil and climatic conditions on the tubers of some plants, whereas those of other plants are free from them. It has been contended that the latter situation is due to the fact that these deformities are often associated with abnormal vine symptoms. Abnormally elongated tubers (pl. 3, D) especially are associated with a condition of the tops that is believed to be due to a virus. The top symptom is known as marginal leaf roll, while the tuber symptom is called spindle tuber. (See Spindle Tuber, p. 49.) Knobbi-

ness and cracking of tubers and an internal browning or spotting are reported as being associated with a vine condition known as yellow dwarf. Further experimental work will have to determine whether these observations and interpretations are correct.

It has been established experimentally that these deformities can be induced by an uneven water supply and by indiscreet use of fertilizers. The symptoms indicate checking of growth, followed by renewal with resultant cracking of the hardened skin or development of knobs at the buds, or elongation. Growth cracks usually are covered with the normal cork layer and therefore are not subject to decay. The knobs and pointed ends, however, especially when they are due to recent excess of water, are immature and tender. Occasionally they contract decay in the soil. (See Jelly End Rots, p. 29.) The knobs are easily broken off, and an easy entrance for rot-producing fungi is thus afforded. In the long Burbank and the Netted Gem varieties in the irrigated sections of Idaho and California second-growth knobs are a common occurrence, and the wounds made when they are accidentally or deliberately broken off open the way for leak and Rhizopus rot. (See Leak, p. 33, and Rhizopus Soft Rots, p. 40.)

In so far as these abnormalities are due to climatic conditions, little can be done to control them. The maintenance of proper soil moisture by frequent cultivation during drought periods is recommended. In the irrigated sections a great deal can be done to prevent second growth by proper handling of the water supply. Care should be taken to avoid breaking off the knobs. (*12; 167, p. 35–36.*)

HOLLOW HEART

Hollow heart is a condition brought about by a too rapid or an irregular rate of growth. It is common in certain large-tubered rapid-growing varieties during wet seasons or in very fertile soils, and is more likely to occur in large than in small tubers. Largeness of tubers, however, is not an infallible sign, because small tubers also may be affected. There is no absolutely reliable external symptom. Hollow heart seriously affects the marketability of potatoes.

Hollow heart consists of more or less irregular cavities of varying size in the center of the tuber. (Pl. 10, A.) The cavity is usually lined by dead tissue that is glassy and brown. Hollow heart can be distinguished from black-heart (see Black-heart, p. 10) in which cavities have developed by the conspicuous discolorations associated with the latter. Hollow heart does not develop in transit or in storage. So far as known, affected tubers are not unsuited for seed purposes.

Control measures involve regulation of the rate of growth of the developing tubers. Seasonal and climatic conditions determine to what extent the grower can force development of his crop by use of fertilizers or by irrigation. His experience will also guide him in selecting varieties that do not grow with abnormal rapidity.

IMMATURITY

Maturity is a very important factor in determining the market quality of tubers. Lack of maturity, or immaturity, is indicated

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A. Hollow heart
B. Growth cracks
C. Mechanical injury and immaturity
D. Second growth, knobbiness, and immaturity

by several characteristics. Mature tubers have a well-developed layer of corky tissue that protects them from excessive drying out, bruising and peeling in handling, and rapid greening in light. Immature tubers can be recognized by excessive wilting and flabbiness (pl. 10, D), by excessive peeling or feathering of the skin, and by the readiness with which they turn green. A considerable amount of this is tolerated in the southern crop and in the early crop of the North, which, however, are marked for immediate consumption. The immature stock is very susceptible to invasion by numerous rot-producing fungi, especially if it is shipped wet and dirty in overheated cars or in tight barrels.

Immature stock should be handled as carefully as possible, if bruising (pl. 10, C), with subsequent drying out and decay, is to be avoided. To avoid loss of water through the unbroken skin, the tubers should be protected from excessively dry air. Greening can be prevented by keeping them out of direct sunlight. The safest way, however, to prevent the ills attendant upon immaturity is to allow the tubers to grow until they develop a normal layer of cork.

INTERNAL BROWN SPOT

The causal complex of internal brown spot has not been determined. The development of this disease in many cases seems to be associated with dry soils or lack of water at some period during growth of the tuber. It seems to be more common and serious in some varieties than in others. It affects the marketability once its presence in a lot is detected. There are no external symptoms, and consequently affected tubers often reach the ultimate consumer before complaint is made.

Internal brown spot is marked by brown or yellow regions of dead tissues scattered throughout the tuber. The affected areas may be small or large, even one-half inch in diameter. (Pl. 6, C.) They may be scarce or numerous, and they are isolated, and not connected as in net necrosis. The color of the lesions is very much like that described under Drought and Heat Necrosis (p. 16). (Pl. 6, D.) Possibly the essential difference is merely one of the extent of the tissues affected.

There are no experimental data available on the basis of which any definite statement as to cause, relation of environmental factors, and control can be made. It is a moot question whether bacteria or fungi are directly involved in the production of the necrotic areas. It is advisable on general principles not to use affected tubers as seed stock. There is no evidence that the disease develops or becomes more serious in transit or in storage. (*9; 12; 167, p. 29.*)

JELLY END ROTS

(*Rhizoctonia solani* Kühn[12] and various Fusarium species)

OCCURRENCE, SYMPTOMS, AND EFFECTS

At times the blackleg organism (see Blackleg, p. 11) produces symptoms that are indistinguishable from jelly end rot, although it appears now that most jelly end rot is induced by *Rhizoctonia*

[12] Syn., *Corticium vagum* Berk. and Curt.

solani. This disease occurs in the Burbank variety in California and in the Netted Gem in Idaho. Its economic significance lies in the fact that it lowers the market value of potatoes or renders them entirely worthless and also opens the way for secondary rot-producing organisms.

The decay usually occurs in those portions of the tuber which are succulent and deficient in starch, such as the pointed ends of the Burbank type and occasionally also in the knobs and at the eye end. (Pl. 9, C.) Before drying out, the affected tissues have a clear, jellylike consistency. Later on, they shrivel down almost to the healthy flesh underneath. The decay develops little or not at all in transit or storage, since affected portions usually dry out immediately after digging.

CAUSAL FACTORS

The fungus *Rhizoctonia solani*, which induces most cases of jelly end rot, overwinters as black scurf on tubers or in the soil, from where it may infect growing tubers. The disease does not develop or spread in transit or in storage, although it opens the way for Fusarium species and bacteria.

(For a detailed discussion of the relation of environmental factors, see Rhizoctonia Black Scurf or Soil Rot, p. 37.)

CONTROL MEASURES

Prevention of jelly end rot depends on the ability of the grower to provide cultural conditions that will prevent the development of pointed tubers. This, in turn, depends upon the proper handling of irrigation water. Tubers that show active stem-end rot in the spring should not be used for seed, because they harbor secondary organisms that can cause losses. (*164*.) (See also Spindle Tuber, p. 49.)

LATE BLIGHT (TUBER ROT)

(*Phytophthora infestans* (Mont.) DBy.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Late blight occurs in all varieties of the potato in varying degree. It affects the leaves, stems, and tubers, causing such heavy losses in the sections best suited for potato culture that it has come to be known as the potato disease.

From the point of view of production, the losses are due to poor germination of affected seed pieces, to destruction of the young sprouts and the green food-making parts of the plant so that poor yields result, and to attack upon tubers so that these either are unmarketable or are marketable only at a reduced price. Losses are not restricted to the field, however, as the disease destroys tubers in transit and in storage. The disease may cause serious damage in any potato-growing section where diseased tubers were planted, whenever very moist, cool weather prevails during the early growing season, followed by moist, moderately warm weather later in the summer. It occurs frequently in severe form in the New England States adjacent to Canada, in the Puget Sound district, and less fre-

A, B, D, E. Late blight tuber rot, shallow lesion type

C, F. Late blight tuber rot, deep soft lesion type in southern stock

quently though often in severe form in New York, New Jersey, Pennsylvania, Ohio, Michigan, Wisconsin, Minnesota, Iowa, in the mountain districts of the Eastern and Western States, and in Florida on the early crops.

In the earliest stages the disease on the tubers is marked by reddish-brown, brown, or purplish-brown spots (pl. 11, D, E, F), which, as development continues, become shrunken and darker colored. In storage stock late-blight tuber rot is moderately dry, firm, or leathery, and does not usually extend more than one-eighth to one-fourth of an inch beneath the skin (pl. 11, A, B); but in newly harvested potatoes, particularly those of the early southern crop, the reddish-brown moist decay often penetrates to a depth of half an inch or more. (Pl. 11, C, F.) The decay may also occur in the tissues at the stem end as a broad brown ring, and in advanced stages the tuber may be converted into a shriveled mass. In newly harvested stock or in stock held in very moist conditions, the white tufts of the fungus often appear at breaks in the skin and at the lenticels.

A serious feature of the disease on tubers is that it opens the way for bacteria and other fungi which rot the tuber much more rapidly than the late-blight fungus. If bacteria get in through diseased spots, a very wet, soft, or slimy and foul-smelling rot results. (See Bacterial Soft Rots, p. 6.) This usually occurs under warm, moist conditions. If other fungi (usually Fusaria) follow it, an odorless rot results which may be wet or dry, depending upon temperature and moisture conditions. (See Fusarium Tuber Rots, p. 21.)

Late blight on the growing plants reduces the yield and under favorable weather conditions may cause the complete loss of entire fields. In the earliest stages it is marked by water-soaked, pale greenish brown spots on the leaves and stems. The spots often begin at the margins of the leaves and eventually cause them to wilt and blacken. In dry weather they shrivel and dry up. When the weather is warm and moist the spots enlarge rapidly, and one spot may kill an entire leaf in one to four days. Under such conditions a white coating of the fungus grows on the lower or upper sides of affected leaves and on the stems, bearing spores in great numbers.

CAUSAL FACTORS

The fungus *Phytophthora infestans*, which induces late blight, overwinters in tubers in storage or in the field. It is not known to overwinter in the soil, although the behavior of the disease at times indicates this as a possibility. Experiments have shown that the organism can spread to the soil and remain parasitic and pathogenic for at least 57 days.

The fungus grows into sprouts from diseased tubers and in this way is carried to the surface of the ground, unless the sprouts are killed before they break through the soil. The fungus may spread in or on the soil for an inch or so if the ground is very wet, or it may grow on the diseased sprout during wet weather and produce great numbers of spores. These may be carried by wind, rain, or insects such as the potato beetle and slugs to near-by healthy plants and start new disease spots. From diseased vines the spores either

fall or are washed to and into the ground by rains, and consequently tubers which are borne within an inch or two of the surface or which are not completely covered with soil become covered with the spores of the fungus. In wet seasons the fungus grows so well in heavy soils and produces its spores so freely on and in the soil that all tubers in the ground may become thoroughly covered with them. The fungus may also grow into the stem end of tubers from a diseased mother plant or from the soil, entering by way of the stolon, but this rarely happens.

Tubers also become thoroughly covered with spores if they are dug before frost while the disease is active in the field or if they are piled in the field and covered with diseased vines. When conditions are favorable for the fungus in transit and storage, spore production and infection may take place there also.

The fungus does not grow if moisture is not abundant or if the temperature is below 40° or above 86° F. It grows best at temperatures between 73° and 80° and when the air or soil or tubers are very moist. Under these conditions it sporulates most abundantly. The spores do not germinate if they are dry or if the temperature is either below 36° or above 86°. They germinate best at about 53°, either in a film of water or in very moist air. Exposure to dry air for six hours kills them. According to laboratory studies conducted with leaves, 50° to 55° are the most favorable temperatures for infection. The optimum temperature for tuber infection has not been determined, but it probably does not vary much from that of leaf infection. Three to twenty-four hours are required for leaf penetration under the most favorable conditions; the period necessary for tuber penetration is not known. It probably is at least as long as the former. Symptoms of the disease do not appear on the leaves for four or five days after the fungus comes in contact with them. In moist air with a temperature between 60° and 75° the disease progresses so rapidly that it may kill the entire plant in one to four days.

The presence of the fungus on a tuber does not necessarily mean that the tuber will become infected. Temperatures below 40° or above 86° F. or dryness will prevent development of the inoculum. On the other hand, since some time must elapse between penetration and the appearance of the first symptoms, it frequently happens that tubers are stored or loaded that look sound but in which the fungus has established itself and which will develop symptoms only if temperature and moisture conditions are favorable. The fungus may lie dormant in tubers for months and then begin to grow as soon as favorable temperature conditions arise. In some experiments it was found that inoculated tubers did not develop the first symptoms for three weeks at 64° to 71° in moist air. Apparently sound tubers taken from a field where the tops were killed by the fungus were stored at 40°, 55°, and 70°, and showed 17, 53, and 19 per cent, respectively, of rotting tubers at the end of two months. In moist air with temperatures above 41° the fungus grows out of diseased tubers and produces spores that can infect near-by tubers with unbroken skin. It has been found that in storage conditions, with moisture and temperatures between 60° and 71°, lesions on tubers may enlarge at the rate of 2 inches in 10 days.

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MARKET DISEASES OF FRUITS AND VEGETABLES

POTATOES[1]

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INTRODUCTION

BOTANICAL RELATIONS

The Solanaceae, or nightshade family, include four species whose vegetative organs or fruits are used as fresh vegetables. The most

[1] This publication is the first in a series designed to aid in the recognition and identification of pathological conditions of economic importance affecting fruits and vegetables in the channels of marketing, with a view to facilitating the market inspection of those food products and reducing and preventing losses from such conditions. The series represents an extended revision and elaboration, with the addition of illustrations, of a preliminary publication entitled "Handbook of the Diseases of Vegetables Occurring Under Market, Storage, and Transit Conditions," by George K. K. Link and Max W. Gardner, which was published by the Department of Agriculture in 1919 especially for the use of the food-products inspectors of the Bureau of Agricultural Economics and was never distributed to the public. In addition to his part in the authorship of the preliminary edition, Doctor Gardner participated in a portion of the revision embodied in the present publication. The material is organized on the basis of the botanical families to which the plants belong, but no botanical system is followed in arranging these families. Practical considerations make it desirable to issue the material in separate sections arranged somewhat in order of the economic importance of the crops. The Host Index of the Fungi of North America, by A. B. Seymour, 1929, is used as a guide to the nomenclature of causal fungi and to the names of authorities therefor. The colored plates are reproduced from colored photographs prepared through the collaboration of Webster Bros., Chicago, Ill., under the direction of George K. K. Link, Max W. Gardner, and Glen B. Ramsey.

important of these, the potato, *Solanum tuberosum* L., is also the most important of all vegetable crops. The others, in the order of their importance, are the tomato, *Lycopersicon esculentum* Mill.; the pepper, *Capsicum annuum* L.; and the eggplant, *Solanum melongena* L. All of these are natives of tropical regions. The eggplant, pepper, and tomato are warm-season plants, while the potato is favored by relatively cool growing conditions.

THE POTATO OF THE MARKET

The potato of the market is the tuber of the herbaceous annual *Solanum tuberosum*, which produces lateral underground stems, the stolons. The tuber is a swelling arising at the end of these stolons, and therefore is a vegetative organ in that it is a fleshy underground stem.

The so-called early potatoes of the market, which come mainly from the Southern States, are tubers that are harvested before the vines have matured and are marketed before they have attained full size and maturity. As a result they have a poorly developed, thin, and tender cork layer, which is easily rubbed off, exposing the tissues beneath, with resultant water loss, discoloration, and increased susceptibility to decay. Because of the poor development of the cork layer and the high water content of the tubers, new potatoes lose water rapidly and excessively, so that they easily lose the firmness which is a desirable market quality.

The so-called late or main-crop potatoes, which come from the Middle Atlantic and Northern States, are tubers which either have been allowed to mature naturally with the maturing vine or have been allowed to lie in the soil for 10 to 14 days to give opportunity for cork development in the case of those vines which have been killed prematurely by frost, destructive animals, or parasites or have been killed intentionally with chemicals to hasten maturity of the tubers.

Such tubers have a well-developed thick and tough cork layer and consequently are suitable for storage. If they are stored at relative humidities of 65 to 95 per cent and at temperatures between 36° and 41° F., there is a relatively short period of high initial water loss, followed by moderate shrinkage during the remainder of the storage period due largely to water loss and to a much lesser extent to digestion and respiration.

Firmness in a potato tuber, therefore, depends to a great extent upon the stage of maturity of the tuber at the time of harvest, the conditions to which it is exposed after harvest and in storage, and the length of the period that has elapsed between harvest and the examination of the tuber.

Texture and color of the cork also determine the market qualities of tubers. In netted varieties good development of netting, and in red varieties good development of color, constitute desirable market qualities.

Different varieties of potatoes are adapted to various culinary uses. Once a potato variety has established itself in the market because of its flavor and culinary qualities, its marketability is determined by many qualities, most of which are considered criteria in determining the grade of tubers. Among these are minimum

sizes for round and long varieties, and absence of anomalies and of pathological conditions or of their effects, which may be directly or indirectly induced by heredity, by mechanical, physical, or chemical factors, by animal (especially insect) depredators, or by parasitic plants or animals.

PATHOLOGICAL CONDITIONS AFFECTING MARKETABILITY

Certain pathological conditions of the vines affect the tubers only indirectly, but result in fewer marketable tubers because of a smaller setting or the development of small, poorly shaped, poorly colored, or flabby tubers. The most important of these pathological conditions are: Anthracnose (*Colletotrichum atramentarium* (B. and Br.) Taubenhaus); Cercospora leaf spot (*Cercospora concors* (Casp.) Sacc.); insect injuries of various kinds induced by the potato leaf hopper (*Empoasca fabae* Harris), the tarnished plant bug (*Lygus pratensis* L.), the Colorado potato beetle (*Leptinotarsa decemlineata* Say), blister beetles (*Epicauta* spp.), the potato aphid (*Illinoia solanifolii* Ashm.), the spinach aphid (*Myzus persicae* Sulz.), the potato psylla (*Paratrioza cockerelli* Sulc.), the potato stalk borer (*Trichobaris trinotata* Say), and the common stalk borer (*Papaipema nebris nitela* Guence); stalk rot (*Sclerotinia sclerotiorum* (Lib.) Massee); tip burn; and the virus disease, mosaic. Tubers from vines affected with mosaic are wholly unsuited for seed purposes.

Certain environmental factors, animal depredators, and parasitic animals and plants not only kill or weaken the vines, thus affecting production directly and the marketability of tubers indirectly, but they also affect tubers directly, destroying them completely, thus reducing production, or lowering their quality so that they become unmarketable. This group, to which belong some of the most serious field diseases of potatoes, includes: Blackleg (*Bacillus phytophthorus* Appel)[2] and *Bacterium solanacearum* (E. F. Smith) E. F. Smith;[3] drought and heat injury; Alternaria tuber rot or early blight (*Alternaria solani* (Ell. and Martin) Jones and Grout); flea-beetle injury (*Epitrix cucumeris* Harris and *E. subcrinita* Leconte); freezing injury; Fusarium tuber rots (*Fusarium* spp.); Fusarium wilts (*Fusarium* spp.); late blight or tuber rot (*Phytophthora infestans* (Mont.) DBy.); potato wart (*Synchytrium endobioticum* (Schilbersky) Percival); powdery scab (*Spongospora subterranea* (Wallr.) Lagerh.); root knot (*Caconema radicicola* (Greef) Cobb); Rhizoctonia scab, stem rot, and root rot (*Rhizoctonia solani* Kühn);[4] Sclerotium rot (*Sclerotium rolfsii* Sacc.); southern bacterial wilt (*Bacterium solanacearum* (E. F. Smith) E. F. Smith; tuber-moth injury (*Phthorimoea operculella* Zeller); Verticillium wilt (*Verticillium albo-atrum* Reinke and Berth.); and violet root rot (*Rhizoctonia crocorum* (P. ex Fr.). Among the virus diseases, leaf roll (net necrosis) and spindle tuber are the only ones that produce direct effects in tubers and are of market significance in table and seed stocks.

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[2] Syn., *Erwinia atroseptica* (Van Hall) Bergey et al.
[3] Syn., *Phytomonas solanaceara* (E. F. Smith) Bergey et al.
[4] Syn., *Corticium vagum* Berk. and Curt.

Certain pathological conditions are restricted entirely to the tubers and are induced either in the field alone or both in the field and after harvest, especially in storage and in transit. This group includes: Armillaria rot (*Armillaria mellea* (Vahl) ex. Fr.); bacterial soft rots (*Bacillus carotovorus* Jones,[5] *B. aroideae* Town.,[6] and *B. mesentericus* Trevisan); black-heart; chemical injury; drought and heat necrosis; enlarged lenticels; fowl injury; greening; growth cracks; hollow heart; insect injuries of various kinds caused by the potato-scab gnat (*Pnyxia scabiei* Hopt.), the seed-corn maggot (*Hylemyia cillicrura* Rondani), the potato seed-piece maggot (*H. trichodactyla* Rondani), white grubs (*Phyllophaga* spp.), and wireworms (several species of the family Elateridae); internal brown spot; leak (*Pythium* spp.); pink rot (*Phytophthora* spp.); Rhizopus rot (*Rhizopus* spp.); rodent injury; scab (*Actinomyces scabies* (Thax.) Güssow); second growth; silver scurf (*Spondylocladium atrovirens* Harz.); and Stysanus rot (*Stysanus stemonitis* Cda.).

Those pathological conditions of vines which do not produce definite anomalies or lesions in tubers are not discussed here.[7]

Plates 1 and 2 afford an example of losses caused by diseases of potatoes in transit to market.

ALTERNARIA TUBER ROT (EARLY BLIGHT)

(*Alternaria solani* (Ell. and Martin) Jones and Grout)[8]

OCCURRENCE, SYMPTOMS, AND EFFECTS

It appears now, contrary to views previously held, that *Alternaria solani* is able to attack tubers in transit and in storage, and consequently is of direct importance in affecting their marketability. So far, the greatest damage has been observed in shipments from Florida, Maine, and Wisconsin, especially in the Spaulding Rose, Early Rose, Green Mountain, Irish Cobbler, and Bliss Triumph varieties.

In addition to the vines and tubers of potato, the fungus attacks other members of the nightshade family, especially eggplant and tomato. In the eggplant, as in the potato, the damage done to the edible part of the plant is for the most part indirect and due to premature death of the foliage, which causes decreased yields. The fungus *Alternaria solani*, like other Alternaria species, attacks plant tissues most readily in the field and after harvest, when they have passed their stage of greatest vigor, especially when they are exposed to unfavorable environmental factors. Potato tubers become infected at harvesting time, and the development of lesions takes place when the tubers are in transit or in storage.

Lesions on the potato tubers involve the cork and cortical parenchyma and are of various sizes and shapes. Generally they are sunken

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[5] Syn., *Erwinia carotovora* (Jones) Holland.

[6] Syn., *Erwinia aroideae* (Town.) Holland.

[7] For information on diseases of potatoes not encountered on the market, but often affecting marketability, the following list of literature citations should be consulted: General diseases—*3, 4, 8, 12, 13, 26, 31, 34, 50, 66, 68, 73, 77, 81, 89, 101, 112, 123, 128, 133, 151, 175, 179;* degeneration diseases—*39, 40, 54, 65, 146, 159, 160;* leaf roll—*1, 2, 6, 20, 41, 67, 119, 122, 127, 192;* mosaic—*30, 71, 131, 138, 157, 161, 172, 178.* Other citations are given at the end of each section relating to a particular disease or condition. Italic numbers in this footnote and those in parentheses in the text refer to Literature Cited, p. 53.

[8] Throughout this publication the scientific name of the organism inducing a disease is shown in parentheses immediately following the heading giving the name of the disease. For a fuller statement regarding the rôle of these organisms, see footnote 9, p. 5.

with regular raised borders, and the cork has a purplish-brown metallic hue. Most lesions are superficial and vary from one-fourth to 1 inch in diameter. (Pl. 3, B.) Externally these spots may appear very similar to those produced by late blight, but internally the lesions are more shallow and are sharply set off from the healthy tissues by a layer of wound cork. This layer of cork makes it possible for portions of the decayed tissue to be lifted out with the point of a knife. A yellowish discoloration of the flesh is sometimes produced just beyond the edge of the lesion, but the decay does not feather out irregularly into the healthy tissue as is common in late blight. (Pl. 3, C.)

The symptoms on the foliage of potato, eggplant, and tomato are very similar. In fact, there is great uniformity in the lesions caused by any Alternaria species on living foliage. Lesions on the leaves are brown to black and usually show a series of concentric rings which produce a target-board effect. There is often a narrow marginal faded zone. The spots are usually oval in shape. On vigorous leaves, however, they may remain small and angular because of limitation by the veins. As the leaf weakens or dies, the spots enlarge. Under conditions favorable for the fungus the lower leaves of plants gradually die and dry up until only a few green spotted leaves remain at the top of the plant. In severe cases lesions develop on the petioles and upper stems of the plant. Sometimes the olivaceous to black fungus, which comes to the surface of the lesions, becomes abundant enough so that it can be seen.

CAUSAL FACTORS [9]

The fungus *Alternaria solani*, which induces this disease, overwinters in the form of mycelium and conidia in plant trash and possibly on tubers as well. In the spring it produces a new crop

[9] In this publication the section Causal Factors under each disease comprises a statement of the complex of circumstances that determine the occurrence of the pathological phenomena under consideration; in other words, a discussion of all known antecedents of the consequences which are recognized as the disease. It is common usage among American plant pathologists to refer to the fungus or bacterium which enters into the condition complex of a pathological phenomenon as "the cause" of the disease. It should be borne in mind, however, that even in a disease induced by a foreign organism many other factors usually determine the occurrence of the disease. Among these are the conditions that govern the occurrence, abundance, and virulence of the foreign organism, foremost among which are (1) such external nonliving environmental factors as temperature, moisture, humidity, and food supply, and (2) the genetic constitution and other internal environmental factors of the organism itself. In addition, occurrence of the disease is conditioned by those factors which determine the resistance and susceptibility of the affected plant, namely, (1) the genetic constitution and other internal environmental factors, such as the age of the plant, and (2) the reaction of the plant during growth, harvest, transit, and storage, to external nonliving environmental factors. Among these the most important are soil, weather, temperature, humidity, chemicals, composition of the surrounding atmosphere, and agents that cause injuries of any sort, especially those that break the protective tissues of the plant. For those pathological phenomena in which no foreign organism enters as a part of the causal complex, either the internal factors of the plant itself or the external nonliving environmental factors, alone or in combination, constitute the sole determining antecedents of the disease. The external nonliving environmental factors play a very important rôle in the pathological phenomena of plants during the growing, harvesting, transporting, and storage of the crop. As in diseases excited by foreign organisms, the significant external nonliving environmental conditions consist of physical, chemical, and mechanical factors, such as temperature, moisture, humidity, chemicals, composition of the atmosphere, and the like. When a specific foreign body, in the form of an organism, such as a bacterium, fungus, nematode, or insect, is the condition or agent which, by virtue of a precedent condition complex, is able to induce or incite a disease, the authors prefer to designate this specific type of disease factor or condition by the term "excitant" rather than by the expressions "the cause" or "causal organism," and to say that it "incites" or "excites" rather than that it "causes" the disease. In conformity with past usage in publications of the United States Department of Agriculture, however, the term "causal organism" is used rather than "excitant" in the body of this publication.

of conidia, which are easily dislodged and readily disseminated by wind, rain, and insects. Frequent rains and dews are necessary for spore production. The germinating conidia enter the tissues by germ tubes through the stomata or directly through the epidermis. The fungus produces spores when the spots are one-eighth to one-sixth inch in diameter, and a single spot may produce 1,500 to 5,000 spores in one season. On artificial media the fungus grows best, and its spores germinate most abundantly at temperatures between 77° and 85° F. There is no growth below 35° or above 112°.

Climate and soil exert a great influence upon development of the disease in the field. The most favorable conditions for the fungus and the disease are periods of high temperatures alternating with moist periods, in combination with a weakened condition of the plant. The fungus may attack both young and old tubers, the lesions becoming visible within about 10 days. Development of decay is very slow at 41° to 45°, most rapid at about 59°, and practically inhibited at 77°.

CONTROL MEASURES

In view of the fact that the parasite overwinters in plant débris, sanitary measures are recommended. These are crop rotation and destruction of the tops of affected vines. It is desirable not to pile vines on or near the tubers at digging time. Spraying with a 5–5–50 Bordeaux mixture, if done thoroughly and regularly (weekly), gives good control of the disease. (*38*, *55*, *56*, *143*, *189*.)

ARMILLARIA ROT

(*Armillaria mellea* (Vahl) ex Fr.)

Armillaria rot has been reported only from the State of Washington, where it is a factor only when potatoes are planted in newly cleared land that is rich in humus.

The fungus *Armillaria mellea*, which induces this rot, furnishes the diagnostic characters of the disease. It forms structures known as rhizomorphs, from which mushroomlike fruiting bodies arise. These, because of their color, have given rise to the name "honey fungus." The chocolate-colored rhizomorphs grow about the roots and penetrate the corky layer of the tuber and, attaching themselves, draw food material from the tuber until it becomes a shriveled mass.

The disease can be avoided by planting corn or summer crops instead of potatoes in newly cleared humus soil until the fungus is starved out. Host resistance appears to be due to physiological antagonism between host and parasite rather than to structural differences. (*11*, *177*.)

BACTERIAL SOFT ROTS (SLIMY SOFT ROT)

(*Bacillus carotovorus* L. R. Jones; *B. aroideae* Towns.; *B. mesentericus* Trevisan)

OCCURRENCE, SYMPTOMS, AND EFFECTS

The term bacterial soft rot is applied to a group of bacterial rots of potato tuber aside from the blackleg rot (see Blackleg), from which

these can not always be differentiated with certainty unless cultural studies are made. This group of rots is also called slimy soft rot.

The decay is typically white to cream colored, with browning at the margins at times. Sometimes, and in some varieties, especially after exposure to air and light, it may become brown to black. The affected tissues are soft and elastic, but usually not decidedly watery. In some types of decay the affected tissues are viscous so that they can be pulled like taffy.[10] The boundary between the soft, disintegrated and the sound, firm tissues is very sharp, and often the outer portions of the tuber are left intact while the inner are completely destroyed. As the soft tissues dry they become slimy, and finally, when completely dry, they may be reduced to mere chalky white masses. (Pl. 4, A, B.)

In nature the bacterial soft rots are often attended by foul odors caused by other than the primary causal organisms. With pure-culture inoculations no odors, or only slight ones, are noted.

Two of the organisms (*Bacillus aroideae* and *B. carotovorus*) that induce bacterial soft rot of potatoes also cause decay of the stems of growing potato plants, producing a soft disintegration of the tissues that may or may not be black.

Bacterial soft rot occurs in potatoes from all sections and probably causes more destruction than do all other potato parasites combined, because it may occur as either a primary or a secondary rot. As a primary disease it is found most abundantly in the early southern immature crop, particularly when badly bruised or skinned potatoes are loaded while hot or wet and shipped with poor ventilation. (Pl. 4, A, B.) As a secondary rot it may follow other diseases or injuries, especially late-blight tuber rot, blackleg, Fusarium tuber rot, leak, brown rot, scald, freezing, and mechanical injury. In the field it often becomes important in water-logged soils as seed-piece decay.

CAUSAL FACTORS

Generally the soft rots of potatoes found in nature are induced by mixtures of one or more of the organisms which can attack live potato tissues, or of one or more of these parasites associated with other bacteria which alone can not attack the live tissues. The presence of the latter tends to modify the symptoms, however. This probably accounts for the fact that the bacterial soft rots produced in the laboratory with pure cultures are practically odorless in contrast with those occurring naturally, which often are vilely odorous. This mixture of organisms may also account for the wide variations in consistency and color of the rots both in nature and in the laboratory. It has been demonstrated experimentally that at least three species or varieties of bacteria isolated from soft-rot lesions on potato tubers can cause rot in pure culture. These are *Bacillus carotovorus*, *B. aroideae*, and *B. mesentericus*. In addition, *B. phytophthorus*, the causal organism of blackleg, and *B. solanisaprus* often induce serious and abundant soft rot of tubers, which can not always be readily differentiated from the other bacterial soft rots. (Pl. 3,

[10] The rot induced by *Bacillus solanisaprus* Harrison (syn., *Erwinia solanisapra* (Harrison) Holland; see Blackleg, p. 11) often exudes a clear liquid. In black rot the early stages are white, but later, especially in light and air, the border turns pink, and then jet black.

A, D.) It has been demonstrated that in addition to potato tubers, *B. aroideae* and *B. carotovorus* attack a wide variety of truck crops—bean, cabbage, carrot, celery, cucumber, eggplant, muskmelon, onion, parsnip, pepper, radish, salsify, tomato, and turnip.

The soft-rot bacteria are normal inhabitants of the soil. Like other soil organisms, their relative abundance can be increased greatly by accumulation of vegetable litter. They are so abundant that there probably are no vegetable crops on which they are not present, owing to washing, splashing, and blowing of the soil. Furthermore, plant tissues are contaminated with these bacteria during harvesting and packing by means of soil, tools, hands, or wash water. Not only are they present as contaminants in the field, but they cause active rot there, as in cabbage, muskmelon, tomato, and in root crops. Much of the rot that develops in transit and in storage has its actual inception in the field.

Since all of these organisms attack potato-tuber tissues through breaks in the protective coverings, practically all invasions of tissue by the soft-rot organisms are through mechanical wounds, the lesions of other diseases, or tissues weakened by mechanical agencies, by freezing, or by aging. Soft rots are the most common invaders of aging or yellowing leafy tissues and of tissues affected with nonparasitic pathological conditions, such as freezing injury, sun scald, and black-heart, or with parasitic diseases, such as bacterial wilt or late blight.

The organisms will invade only moist and turgid tissues and can attack dead or injured tissues only when they are wet. Breaks in the tissues or moistened surfaces provide them with the water and food they need to get started and to carry on growth and reproduction. The rot may be spread in transit or storage by contact or by oozing and dripping from the decayed tissues. There probably is much less actual spread of this decay in transit than of many fungus rots, because there is no aerial mycelium. *Bacillus carotovorus* is particularly sensitive to desiccation and to direct sunlight.

Wet weather favors the disease in the field directly and indirectly. It brings about a multiplication of the organism, disseminates it, moistens plant tissues, makes the plant tissues more turgid, and often suffuses them with water so that they break or crack.

The temperature relations of *Bacillus aroideae*, *B. carotovorus*, and *B. mesentericus* are not identical; consequently, temperature to a great extent will determine which organism will invade a given lot of potatoes. *B. carotovorus* attacks potatoes at temperatures ranging from 46.5° to 92° F., but causes comparatively little rot outside the range 70° to 84°, most rot occurring between 70° and 77°; *B. aroideae* invades tubers beginning at 52° and is a very active producer of rot at temperatures between 68° and 102°; whereas *B. mesentericus* invades tubers only slightly at 53.5° but is active between 68° and 107.5°. At 98° *B. carotovorus* no longer induces rot, while *B. aroideae* and *B. mesentericus* induce very active rot. At 102° *B. aroideae* no longer causes rot, while *B. mesentericus* causes rot at the upper limit of temperatures tolerated by potato tissues. Consequently, *B. carotovorus* is a potential danger in cool storage, while the other organisms are most significant in tubers that become heated in the field, in transit, or in storage.

Bacillus phytophthorus, the causal agent of blackleg and of a soft tuber rot, invades tubers at temperatures between 35.5° and 92°, being most destructive between 42° and 84°. *B. solanisaprus* induces slight rot at 37.5° and active rot beginning at 68° and extending above 89.5°. Its lower and upper temperature limits have not been determined. At a temperature around 87° all the organisms cause active rot.

It has been experimentally demonstrated that in very dry air there is little infection by any of these organisms through either surface or deep wounds, and that in practically saturated air each of the organisms can infect through a surface wound or a deep wound at temperatures of 68° to 77°. Relative humidities below 80 per cent retard progress of the rot even after it has become established by way of deep wounds.

Striking differences in resistance and susceptibility to the rot have been established experimentally for tubers of different potato varieties. Ten varieties tested showed the following ranking on the basis of increasing resistance to bacterial soft rots: Rural New Yorker, Russet Rural, Burbank, Irish Cobbler, Green Mountain, Triumph, Early Ohio, Early Rose, McCormick, and Spaulding Rose. Burbank, Early Ohio, Spaulding Rose, and Green Mountain, after prolonged storage, are more subject to rot induced by the blackleg organism than to the soft rots induced by *Bacillus aroideae*, *B. carotovorus*, and *B. mesentericus*. Spaulding Rose is resistant to all rots save that induced by the blackleg organism.

CONTROL MEASURES

As in many other storage rots, the control of bacterial soft rots begins in the field. One step is the prevention of diseases of the growing or maturing crop, because these increase the reservoir of infection of the bacterial soft rots by providing dying or dead plant material. Furthermore, field diseases produce lesions in tubers through which soft-rot infection may occur, as in late blight, brown rot, or blackleg.

Crop rotation will not only aid in the control of certain field diseases, but will also lessen the reservoir of soft-rot bacteria in the soil. The reservoir of contamination in the field may also be greatly reduced by a reasonable degree of sanitation in the disposal of culls, trash, and litter. In packing, rotting tubers should be sorted out, and these and other rotting vegetables should not be thrown on manure or compost heaps to be eventually returned to the field. In fact, every precaution should be taken to guard against the use of contaminated manure or compost. Proper handling during harvest to avoid all unnecessary wounds, cuts, bruises, sun scald, and freezing injury is of primary importance in the prevention of soft rot. The potato, like many root crops, may profitably be allowed to dry before being stored or shipped. This will kill the soft-rot bacteria on the surface of the tubers and allow all surface wounds to become dry.

Ventilation and low temperature during transit and storage are, of course, most important factors in the control of this rot. Ventilation, by reducing the relative humidity and preventing condensation

of moisture on surfaces, renders the tissues less turgid and so less susceptible to infection. Furthermore, ventilation disposes of the products of respiration, including heat, and prevents black-heart, which predisposes potatoes to soft-rot infection. Storage pits, bins, and houses should be properly constructed so as to provide for the necessary ventilation. Where actual refrigeration is impossible, much can be accomplished by properly ventilated cool storage and transit conditions. Temperature control during storage and transit is probably relied upon to a greater extent than any other factor for the prevention of soft rot. Soft rot will not make any progress in refrigerator cars after the products have once become thoroughly cooled.

Because of the offensiveness of the bacterial soft rot, salvage and reconditioning of partially affected lots of potatoes is less feasible than it is with the fungous rots. However, by careful sorting apparently worthless shipments may be salvaged provided whatever wounded stock is obtained can be used at once or exposed to dry air, especially in direct sunlight. (*17*, *21*, *59*, *72*, *82*, *89*, *92*, *104*, *115*, *167*.)

BLACK-HEART

Black-heart is the result of abnormal conditions in and subsequent death of the tissues of the potato tuber, with definite post-mortem changes, induced by a deficiency of oxygen. It may be brought about at any temperature by an absolute lack of oxygen, as a result of inadequate aeration, or in full supply of air if the temperature is so high that it causes a rate of oxygen consumption by the tissues in excess of that supplied by normal air through the outer tuber tissues. These conditions result in death of the cells, the most secluded ones of the pith and phloem parenchyma dying first. Death of the cells is followed by discolorations, the ultimate jet black noted in affected tubers having given rise to the name of black-heart. The discolorations are the result of unregulated interaction among various constituents of the normal cell and free oxygen. The enzyme tyrosinase acting upon certain pigment-forming substances in the presence of free oxygen leads to the development of a group of substances, among them melanin, which give the pink, brown, and black colors characteristic of the various stages of black heart. (Pl. 5, C, D, E.) It should be borne in mind that the colors noted in black-heart can be found quite generally in killed potato tissues. There is some suggestion of them in freezing injury (see p. 17) and almost a duplication of them in leak (p. 33), pink rot (p. 35), and in some types of Fusarium tuber rot (p. 21). They are noted also in "scalded" tissues. (See Scald, p. 46.)

Black-heart is not known to occur in growing tubers, except occasionally in flooded soils. It occurs in the field, however, after digging, when tubers are allowed to lie in or on hot soil exposed to the sun. It occurs in transit when the temperature in stove-heated cars is allowed to go over 90° F. It occurs also in storage houses and pits if the piles of tubers are made so large that the interior can not be well aerated. Under those conditions it may even occur in refrigerated rooms, especially if the tubers are hot and wet when placed in storage.

The symptoms of black-heart vary, depending upon whether the tubers were exposed to high temperatures with a normal air supply, or to high, low, or normal temperatures with an insufficient air supply. In the former case no external symptoms develop; in the latter, both external and internal symptoms appear. The killed tissues are easily invaded by bacteria and fungi, which may obscure typical black-heart symptoms by causing various forms of watery or slimy decay. The external symptoms of black-heart are moist areas on the surface which may be purplish at first but turn brown or black within a short time. The internal symptoms are a dark-gray to purplish or inky-black discoloration. Injured tissues are of normal color when freshly cut soon after the injury of the tuber, but shortly after the air strikes them they turn pink, then gray or purplish, and finally jet black. (Pl. 5, C, D, E.) Sometimes all the colors except pink are found simultaneously in the same tuber; at other times only gray or brown are found, which is the case when tubers are heated above 130° F., or when they are deprived of oxygen for considerable periods after death, as in water-logged soils or in flooded pits. The colors usually are sharply set off from the healthy tissues. The affected tissues are firm and even leathery if they have dried a little—quite unlike those affected with leak, which frequently show colors similar to those of black-heart. When the discoloration is due primarily to insufficient oxygen it is restricted to the heart of the tuber, but it may radiate to the exterior as well. When one-sided exposure to high temperature is involved, as in a stove-heated car or in exposure to the sun in the field, the discoloration may affect only the exposed side of the tuber. (Pl. 5, D.) The discolored regions may appear in zones in the outer parts of the tuber and be absent or less evident in the center. In advanced stages the tissues dry out and cavities result. (Pl. 5, E.)

Since tubers will not usually develop black-heart at temperatures below 95° F. if given a good supply of air, control of the injury involves storage at temperatures below 95° with good ventilation. In stove-heated cars or storage places the tubers near the stove should be protected by tin sheeting. The temperature in heated cars should not be allowed to go above 60° or 70°. To prevent a shortage of oxygen, tubers should not be left long in hot, light soils after the vines are dead or be left lying near or on the surface after digging during hot weather. Flooding fields or packing hot, wet, or soil-covered tubers in closed containers, such as barrels, should be guarded against, since these methods may lead to asphyxiation. (*14*, *15*, *16*, *29*, *174*.)

BLACKLEG (TUBER ROT)

(*Bacillus phytophthorus* Appel)

OCCURRENCE, SYMPTOMS, AND EFFECTS

The disease known as blackleg of potatoes occurs in practically all parts of the United States and is most abundant in wet, warm seasons. It affects all parts of the plant and is of importance not only because it kills the growing plant and thus reduces the yield, but also because it affects the tubers, rotting them in the field, in transit, and in storage, and opening the way for other bacterial soft rots. (See Bacterial Soft Rots, p. 6.)

The disease derives its name from the fact that the lower portions of affected stems are characterized by relatively dry jet-black lesions. The tops of affected plants have an upright, staring appearance, due to a hardening of the vascular bundles, and generally are yellowish. If the plant is attacked early it generally is dwarfed and dies without any tuber production. If infection takes place later, a crop of tubers may be produced, some of which may become infected through the stolon from the mother plant. In irrigated sections infection often proceeds from the crown of the plant. In these cases the pith of the plant is destroyed, and dead black areas often run far up into the stems, extending to even the outermost leaves. Infection can also proceed through the lenticels, especially in wet soils.

When new tubers contract the disease from the mother plant through the stolons, the stem end of such tubers is the first to show symptoms. The early symptoms are browning and softening of the tissues about the vascular ring. In very moist places, especially under irrigation conditions in the West, tubers also become infected through the lenticels by contact with diseased ones. Some spread of the inoculum may take place in storage by this method. The decay often proceeds into the pith, destroying it completely and leaving a hollow with a black and slimy lining. (Pl. 3, A.) In the long western varieties the decay may develop as a stem-end rot, producing a shriveled, shrunken external appearance. (Pl. 3, D.) Often the entire tuber is converted into a pulp which in freshly decaying tissues may be white or only slightly colored, and cheesy or buttery in consistency. (Pl. 4, B.) Gradually this pulp becomes slimy and black as decomposition progresses. (Pl. 3, A. D.)

In typical tuber rot induced by *Bacillus phytophthorus* the decaying tissues at first are white or yellowish, but later turn pink and jet black along the border. Often there is a fishy odor. These characteristics generally serve to differentiate this rot from the bacterial soft rots. (See p. 6.) The decay induced by *Bacillus solanisaprus* (an organism closely related to *B. phytophthorus*) is soft and white at first, and either remains so or becomes slightly brown upon exposure to air. There is no border which turns from white to pink to jet black, and there is no disagreeable odor. These characteristics serve to differentiate this rot from typical blackleg rot due to *B. phytophthorus*. When *B. solanisaprus* attacks live stems it does not produce black lesions as does *B. phytophthorus*, but green-colored ones if it produces rot at all. The tissues decayed by *B. solanisaprus* often exude a clear liquid, and this characteristic may serve to distinguish it from the bacterial soft rots caused by *B. aroideae*, *B. carotovorus*, and *B. messentericus*. *Bacillus phytophthorus* and *B. solanisaprus* play an important rôle in tuber rots under cool-storage conditions.

In addition to the potato tuber and potato stems, *B. phytophthorus* rots the following vegetables: Bean, cabbage, carrot, tomato, and turnip.

CAUSAL FACTORS

It is well established that typical blackleg is induced by the bacterium *Bacillus phyiophthorus*, and it is fairly well established that

the closely related organism *B. solanisaprus* induces similar symptoms in potatoes. It has been shown that *B. phytophthorus* can overwinter in tubers in storage and that it is carried into the field by them. The inoculum is frequently spread in the preparation of tubers for planting by being carried over from infected to sound tubers. This occurs more commonly than is surmised, because infected tubers are not necessarily discolored or decayed. Whether the organism overwinters in the soil and is a factor in causing infection is a moot question. Observations and experiments in Minnesota and in western potato sections indicate that this is possible, while experiments in the East indicate that the organism does not overwinter in the soil, except in so far as it is associated with the seed-corn maggot. It is well known that the young sprouts become infected from diseased seed pieces, which invariably decay into foul-smelling masses of pulp. Since the organism can enter through lenticels, in wet soils the decay spreads from tuber to tuber by contact, and there is every indication that irrigation water carries the organism from hill to hill. An abundance of moisture is necessary for the development of the disease in the field. In addition, moderately high temperatures are essential.

The seed-corn maggot (*Hylemyia cilicrura* Rondani) and the potato-seed maggot (*H. trichodactyla*) play an important part in infection of cut seed pieces in the air and in the soil. If cut pieces are placed in excessively wet soil there is poor development or no development of wound cork, and the tissues are readily affected by rots of diverse causation. In the soil the cut surfaces and the decaying tissues of such seed pieces attract the yellowish-white legless maggots of these insects, if they are not already present, and they burrow over the surface and through the seed pieces. The surface of the eggs of these insects is often infested with the blackleg organism, which eventually gets into the intestinal tract of the larvæ and is thus carried into the tuber tissues by the maggots.

The rôle of temperatures and humidities in tuber decay induced by these organisms and experimental data in relation to the resistance and susceptibility of tubers of various potato varieties to *Bacillus phytophthorus* are discussed under Bacterial Soft Rots (p. 6).

CONTROL MEASURES

The only method of control in the field is to rogue all diseased plants as soon as they appear. In the eastern potato sections the most severe attack of the disease occurs in the early part of the growing season. In the irrigated sections of the West the new infections appear throughout the growing season, practically up to digging time. After the harvest all diseased tubers should be discarded, because they are likely to decay and spread the infection to their neighbors. Tubers from infested hills should not be used for seed even when they appear sound. Discolored or decayed tubers, of course, should not be used. In case one has cut into an infected tuber, the knife should be disinfected before further use. Treatment with either formaldehyde or corrosive sublimate solution is regarded as advisable to disinfect the surfaces of tubers that may have become contaminated with the organism through contact with

diseased tubers in handling or in storage. For control of the tuber rot in transit and storage, the discussion of bacterial soft rot (p. 6) should be consulted. (*7*, *18*, *19*, *21*, *70*, *79*, *84*, *85*, *86*, *113*, *115*, *129*, *130*, *142*, *150*, *166*, *171*.)

BROWN ROT (SOUTHERN BACTERIAL WILT)

(*Bacterium solanacearum* (E. F. Smith) E. F. Smith)

OCCURRENCE, SYMPTOMS, AND EFFECTS

In addition to the potato, all varieties of which are susceptible, brown rot or bacterial wilt occurs on eggplant, pepper, tomato, tobacco, jimson weed, black nightshade, peanut, and other plants. It apparently does not occur in the northern half of the United States. In the South it seems to be most serious in the first crops planted on virgin soil.

From the point of view of production, the losses are due to early death of plants, so that they produce either a diminished crop or none. The quality of fruit produced by sick plants is likely to be inferior, owing to underdevelopment. Losses are not restricted to the field, however. In the potato, affected tubers may merely be discolored and as a result bring a reduced price, or they may be invaded by secondary organisms and decay. (See Bacterial Soft Rots, p. 6.)

Brown rot or bacterial wilt occurs most frequently in the warmer sections of the United States and for this reason is often referred to as southern bacterial wilt. The parasite seems to be native to some of the virgin soils of the South Atlantic and Gulf States. Consequently the disease is important, especially in crops grown on new land in Alabama, Florida, Georgia, Louisiana, Mississippi, North Carolina, South Carolina, and Virginia. It is not limited to the United States, however, for reports of serious losses come from the Tropics.

Under transit conditions brown rot is a common and frequently a serious factor in shipments of the early or new crop from States on the Gulf and Atlantic seaboard. Since practically none of this crop goes into storage, the disease is of little importance as a storage rot. On the tuber the external symptoms of brown rot may appear as a slight depression at the point of attachment to the stolon, or as a grayish-brown discolored patch on the surface. (Pl. 4, D, E.) Sometimes no external symptoms develop. Upon cutting across infected tubers a moist, brown discoloration and a slight softening of the water-conducting tissues are seen. Frequently, though not invariably, yellowish-white, sticky globules of bacteria ooze out from the discolored tissues. (Pl. 4, C, E.) At this stage there is no odor. Later, as the bacteria work toward the center of the tuber, the entire interior becomes soft and brown, while a thin crust of tissues holds the tuber together. If the rot reaches this advanced stage while the tuber is still in the soil the shell is usually broken when the tubers are dug, and the bacteria-filled contents are left in the soil. If the rot reaches this stage during the period of transit or while the potatoes are in storage, neighboring tubers are likely to become smeared with the rotten contents. In the later stages of the disease other organisms gain entrance (pl. 4, D), the tissues are converted into a creamy consistency, and an indescribably foul odor results.

In the growing plant the outstanding symptom is wilting, which appears rather suddenly. At first it may be restricted to a single shoot, but generally it ultimately affects the entire plant. Sometimes the foliage yellows before it wilts, but if the onset of the disease is sudden, wilting occurs without previous yellowing. The stems shrivel if they are not very woody, usually turning yellowish green at first and finally becoming brown or black. A cross section of the stem or leaf petiole shows brown dots or a brown ring, and the discolored vascular tissues often show through the green parts of the stem and petioles as long, dark streaks. Closer examination shows that the bundles are filled with bacteria, which may ooze out upon cutting. The presence of the bacteria in the water ducts causes clogging, and if the bacteria occur suddenly in great numbers the plant wilts quickly without becoming yellow.

CAUSAL FACTORS

The bacterium that induces brown rot (*Bacterium solanacearum*) seems to occur in certain virgin soils of the South. This statement is based on observations to the effect that the disease occurs in potato crops and other solanaceous crops grown from disease-free seed, on land that had never before produced potatoes or even members of the family. In fact the disease is most severe in potato crops grown on new soil and diminishes in frequency and severity the longer the soils are cultivated and cropped. The bacteria in the soil attack the roots and underground stems of plants and thus gain entrance to the conducting tissues, along which they travel or are carried to all parts of the plant. If the soil is free from the organism, the latter may be carried into a field by insects from diseased crops in near-by fields. Once a single plant becomes diseased in a field, neighboring plants are in danger, unless leaf-cutting insects are held in check. It has been reported that when the Colorado beetle was allowed to feed on diseased plants and was then removed to healthy plants to feed on the tops, symptoms of the disease appeared in these plants in seven to nine days. It has not been demonstrated whether other insects, such as flea beetles or blister beetles, may spread the disease. Neither is it known whether the organism can enter through the unbroken surface of the plant.

Tubers get the disease from the infected plant through the water-conducting tissues. In a greenhouse experiment, three weeks after symptoms appeared in the tops the tubers were found to be entirely or partly rotted. There is no spread of brown rot from tuber to tuber in the marketing process, but it is probably true that the bacterial rot which follows does spread by contact. (See Bacterial Soft Rots, p. 6.)

Warm, moist weather favors the development and spread of the disease. The parasite grows well at temperatures ranging from 77° to 97° F. As the temperature is reduced the rate of growth is retarded, and growth is practically stopped at 55°. The disease consequently can not develop at temperatures below 55°, and it develops most rapidly between 77° and 97°. It seems to progress most rapidly in young plants. Unless infected tubers are held below 55° in transit, they are likely to decay.

CONTROL MEASURES

Satisfactory control measures can hardly be recommended until more is known about this disease. The most serious cases apparently occur on virgin soils, and when planting under these conditions growers should realize the risk. The question as to whether or not continuous potato cropping on the same fields tends to increase the chances of disease needs investigation. Some Florida growers have reported that with continuous cropping the danger of brown rot diminishes.

Tubers that are known to be diseased should not be stored or shipped, even though the trouble may be only in the initial stages, for such potatoes rot very quickly at temperatures above 55° F. As it is practically impossible to discover and remove all tubers that are in the early stages of the disease, potatoes from affected fields should be used immediately or shipped to near-by consuming centers, or else stored under cool, dry conditions, with good ventilation. (*105*, *169*, *170*, *173*.)

CHEMICAL (SALT) INJURY

Potatoes suffer from an injury caused by common salt, and some other chemical salts, such as fertilizers, produce the same effect. Injury is also caused by asphalt, coal tar, and oil products. In all of these mere contact with the chemical apparently is not sufficient to induce the injury. If the substances are ground into the tissues of the tuber by pressure or rubbing, however, the characteristic symptoms are developed. This occurs most commonly when potatoes in storage or in transit are piled on floors or in contact with walls that are impregnated with the chemicals enumerated. Affected tubers are unmarketable, and through the decay that often sets in they become a menace to sound tubers.

Usually the side of the potato in contact with the chemical is flattened (pl. 5, A, B), and the injured tissues are soft and flabby and at times semiwatery. Sometimes the chemical can be tasted within the killed tissues. The injured tissues frequently are firm and leathery, especially if they are not invaded by microorganisms. When the injury is first done the tissues are not discolored, but after a short exposure to the air they become brown to black and dry out. (Pl. 5, A, B.) The exterior of the killed area is usually black.

Control of this injury consists in keeping potato tubers from coming into contact with the chemical.[11] (*44*, *167*.)

DROUGHT AND HEAT NECROSIS

As the name implies, the pathological condition designated as drought and heat necrosis is considered to be due to drought or heat or a combination of both. Little more can be said about the causal complex until experimental work is done. The condition affects the tubers and lowers their market value.

This pathological condition has been noted in tubers grown in light, hot soils in the hot potato-growing sections of the United

[11] LUSK, J. P. INVESTIGATIONS ON SALT INJURY OF POTATOES. 1926. [Master's thesis. University of Chicago, Department of Botany.]

States. It has been noted especially in the early crop grown in the volcanic ash soils of Idaho, and it seems to occur in tubers that are allowed to lie in hot soils when the vines suffer from lack of water and begin to die. It is marked by the death and a golden yellow to brown discoloration of the vascular tissues and of the parenchyma immediately surrounding them. (Pl. 6, D.) The discoloration is most pronounced in the ring tissues, at either the stem or the bud end, and in the outer phloem tissues. It thus differs from net necrosis (pl. 6, E), which it somewhat resembles both in color and in distribution of the necrotic tissues. Discoloration at first is restricted to the vascular and immediately adjoining elements, but after a time it spreads slightly to surrounding parenchyma, and the color changes from golden yellow to a light or dark brown. Sometimes in light-skinned varieties the discolored tissues give a darkened appearance to restricted areas of the surface. Upon cutting, it is found that the discolorations are not due to a solid mass of tissue as they appear to be, but rather to the discolored strands, which impart a dark hue to the tissues overlying them. (Pl. 6, D.)

Control of the disease involves keeping the soil moist, cool, and shaded and in digging the tubers as soon as the vines begin to die, if the soil is light and the weather hot. (*167.*)

ENLARGED LENTICELS

Enlarged lenticels affect the marketability of potatoes, but they do not predispose them to decay, nor do they impair their food value. Under normal soil and atmospheric conditions the lenticel cells are dormant. In very wet or water-logged soil and in very humid atmosphere these cells enlarge and divide. If this growth continues long enough, a mass of cells is formed which protrudes above the tuber surfaces. (Pl. 5, G.) At first these cells are quite soft, whitish in color, and easily rubbed off; later they harden and become brown and corky. (*167, p. 22.*)

FREEZING INJURY

OCCURRENCE, SYMPTOMS, AND EFFECTS

Upon thawing, tubers frozen to death may show freezing injury so plainly externally that they are unsalable and unfit for table purposes. On the other hand, the injury from less severe freezing may be so slight that it is not apparent externally and is revealed only when the potato is cut, perhaps several hours after the exposure to low temperatures. This condition is commonly referred to as frost necrosis. Even though the nonaffected areas, which may be considerable, are fit for food, tubers so affected are not desirable for table purposes, because they are unsightly when served, or because the removal of discolored areas involves an inordinate waste of time.

Storage or shipment of frozen tubers or tubers affected with freezing injury involves hazards of total loss through secondary decays or at least through excessive wrinkling and shriveling due to drying out. The use of affected stock for seed purposes may lead to weak plants or to poor stands because of seed-piece decay.

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The symptoms of freezing injury vary, depending upon what kind of tissues are killed, the amount of tissues involved, and the consistency and color of the affected regions. Which of the symptom complexes will develop depends upon whether the injury is due to (1) long exposure to temperatures just below the freezing point, or to moderately long exposures to very low temperatures, or whether it is due to (2) a moderately short exposure to temperatures just at the freezing point or short exposures to temperatures far below the freezing point. In both of the former cases most or often all of the tissues may be killed, and both external and internal symptoms may appear shortly after thawing. In the latter case only the most susceptible tissues are killed, and no external symptoms are apparent for weeks or months. Short exposures generally produce slight injury of the net or ring types, or a combination of these; longer exposures the blotch type, often in combination with the net and ring types. The restricted or limited type of injury is known as freezing or frost necrosis.

Frozen potato tissue, before thawing, no longer possesses the crispness of the normal tissue. It is abnormally firm, looks dull, and does not cut readily or with snap. This is due to the presence of ice. When ice occurs in only part of the tuber, its presence at times can be detected by a crunching sound if, while held to the ear, the tuber is pressed between the fingers.

Tubers that are frozen solid collapse promptly upon thawing, becoming soft and watery. (Pl. 6, A.) Their wet surfaces make detection and sorting out easy. The presence of moisture on a tuber is not necessarily a sign of freezing injury, however, since cold tubers when brought into moist air with a temperature higher than their own very readily condense water from the air on their surfaces. Sometimes if only a portion of a tuber is exposed to freezing temperature the exposed portion is frozen to death and upon thawing becomes turgid and blisterlike, and the skin frequently discolors. If tissues that have been frozen to death are cut shortly after thawing, they may appear dull and colorless at first. Upon exposure to the air they frequently pass promptly through pink and red color stages and ultimately become brown, gray, or an inky black. (See Blackheart, p. 10.) The rate of development of these colors depends upon the temperature of the air. At temperatures below 75° F. several hours may be required, while only one-half to one hour may suffice at higher temperatures. The occurrence of these color changes is also determined by the extent to which air reaches the affected tissues.

When extensive areas of tissues are killed by freezing, they usually become infected with bacteria, which cause foul-smelling, slimy, or sticky rots if thawing takes place in a warm, humid atmosphere. They dry down to a mealy or tough, leathery, granular, chalky mass of starch and tissue remnants if they thaw in cold or dry air. Tubers that have only one side frozen frequently have the killed portion sharply set off from the nonaffected area by a purplish or ultimately brown line of corky tissues. (Pl. 6, A.) Often infection by Fusarium species sets in before the unaffected cells are sealed off by the corky layer.

It is generally known from experience with potatoes in the field, in storage, and in transit, and from experiments, that when a lot of

tubers has been exposed to low temperatures for a long enough time to freeze some solid, there frequently are others that do not freeze solid and subsequently collapse upon thawing, but which seem to be unaffected so far as external appearances indicate. If such tubers are cut they may show the various types of symptoms known as freezing or frost necrosis. (Pl. 6, B.)

The mildest type of freezing necrosis is one marked by tissues that are drier than surrounding tissues and that have a grayish-white tint. When the injured tissues are first cut soon after thawing, they may turn red in a short time and finally become brown. In uncut tubers, even after a long time, the only sign of injury is a grayish, dull, dry, collapsed appearance of some of the tissues.

Generally, however, freezing necrosis is marked by decided discolorations. These may not be apparent immediately after thawing, but they usually set in after five or six hours. The color changes usually from dull white to pink or brick red and finally to gray, brown, or black.

There are several types of discoloration. One is the ring type (pl. 6, B), which is limited to the vascular ring and immediately adjoining tissues and is characterized by pronounced blackening. Another is the net type, which is marked by more or less blackening of the vascular ring and the finer strands which extend from it into the inner phloem parenchyma and outer phloem parenchyma. (Pl. 6, B.) Both types are frequently restricted to the stem end. Finally, there is a blotch type, marked by irregular patches ranging in color from an opaque gray or blue to sooty black. These patches occur everywhere in the tuber, though most generally in the vascular ring and cortex. When these blotches are in the cortex they may be apparent externally in clean tubers with white skins. This is the only type of freezing necrosis that may be visible externally. If tubers affected with any or all of these types of freezing necrosis are held in storage, they generally shrivel or wilt more than nonaffected tubers. Excessive shriveling alone, however, can not be relied upon as a sign of freezing necrosis.

It is popularly believed that the symptoms here described as freezing necrosis are not due to freezing but to chilling, since freezing, it is contended, is always followed by complete breakdown after thawing. This is not the case. Freezing necrosis is as truly a freezing injury as the complete type and does not occur unless ice is formed in the tissues. The difference lies in the fact that not all cells of the tuber, but only those that become discolored, have been killed.

If tubers are exposed to low temperatures, but not low enough to cause ice formation, sugars accumulate, and the tubers become sweet. This sweetness disappears if the tubers are kept at temperatures above 40° F. Frozen tissues are no sweeter than uninjured ones. Sweetness of tubers, therefore, is not a sign of freezing, as is popularly believed.

Experiments show that the various tissues of the potato have a varying susceptibility to freezing. The vascular tissues are most easily killed, and therefore, with a given temperature and exposure, they frequently are injured, while the other tissues either do not form ice or thaw without visible injury. The vascular tissues of the stem end of the tuber are also more susceptible than those of the bud end.

Consequently, freezing necrosis usually proceeds from the vascular tissues and begins at the stem end.

Potato tubers will not freeze at 32° F., the freezing point of pure water, because the sap or juice of potatoes is not pure water but a solution of salts, sugars, and other more complex materials. The expressed sap freezes at about 30.8°. The freezing points of the tissue is even lower. This is probably due to the influence of the living cells and the capillary action of the intercellular spaces.

The average freezing point of 18 varieties of potatoes has been found to be 28.9° F. Varieties belonging to the early or midseason groups have a higher freezing point than those of the late-season groups, and it has been observed that the freezing point of a given variety falls as the season progresses. The freezing point also varies somewhat with the temperature at which the stock has been held. Generally speaking, a lot of potatoes stored at 32° for several weeks will stand about 1 degree lower temperature without freezing than a similar lot that has been stored at 50°.

It is impossible to forecast the critical temperature for an individual tuber and the time of exposure to low temperatures requisite for injury, because there is great variation in the individual susceptibility of tubers to freezing. Even though the critical low temperature—that is, the freezing temperature—for living potato tissue lies between 28° and 30° F., the temperature of tubers can fall below this point without ice formation, owing to the phenomenon known as undercooling. Associated with this is the phenomenon termed "inoculation," which consists of an instantaneous formation of ice and a sudden rise of temperature in the undercooled tissues to their true freezing point, following a mechanical disturbance such as jarring or a sharp blow.

The degree to which a tuber tissue will undercool depends among other factors upon the air temperature, the time during which it is exposed to low temperatures, and the rate at which undercooling takes place. The faster the temperature falls the less undercooling is tolerated by the tissues. Tubers exposed to air whose temperature dropped slowly to 23° F. undercooled to 23.6° and 23.1° after 165 and 174 minutes, respectively, before freezing, while others exposed at once to the same temperature undercooled only to 24.5° in 73 minutes. Tubers exposed to a temperature of 8.7° froze in 6 minutes, their temperature falling to 23°, while others, exposed to 15.8°, froze in 12.5 minutes.

Tubers whose surfaces are wet or which have newly made wet wounds frequently undercool only slightly if at all. That mechanical disturbance of undercooled tubers leads to freezing injury is proved by the following experiments: Tubers kept for varying lengths of time at 26°, 27°, and 28° F., respectively, were dropped through distances varying from 1 to 4 feet. They showed freezing necrosis. Others treated identically but dropped only 2 to 6 inches showed no injury. However, when struck with a pencil they froze. Continuous bouncing through a distance of 1 inch for 10 seconds produced similar results. Tubers kept at 28° were rolled 30 and 50 feet in a bag. Many of them showed injury. In all cases the controls—that is, tubers kept at the same temperature but not subjected to dropping, striking, or rolling—remained uninjured. In this experiment varietal and individual susceptibility again were apparent.

Frozen tubers should be sorted out as much as possible, since they are subject to severe drying and shriveling as well as to secondary rots, which may become a menace to the unaffected tubers. It is, of course, impossible to sort out tubers that do not show any external symptoms. In case there is reason to suspect freezing injury in a lot of potatoes it is advisable to cut some tubers to determine the presence or the extent of the trouble.

If an affected lot is sold, either a rejection of the entire lot or a demand for an allowance may be expected, since affected stock is not desirable for table, seed, or storage purposes. Sometimes an affected lot reaches the consumer before any difficulty is experienced, and at times it may even go into consumption without complaint. Affected tubers can be used for stock feed and the preparation of potato starch.

CONTROL MEASURES

The prevention of freezing injury involves the protection of tubers against exposure to temperatures lower than 30° F.

Tubers should be dug before severe freezing temperatures set in. If they can not be removed to shelter at the onset of freezing weather, or if they are loaded or unloaded during cold weather, they should be protected by straw, canvas, sacks, and the like. If they are pitted, the covering should be sufficient to prevent internal temperatures below 30° F.

In cars or storage places when the temperature is likely to fall below 30° F. artificial heating must be resorted to. When this is the case it is essential that the potatoes be set away from the walls and above the floor by a false floor, so as to permit ready movement of air. (*76*, *78*, *98*, *193*, *195*, *196*.)

FUSARIUM TUBER ROTS

(*Fusarium* spp., primarily *F. trichothecioides* Wr., *F. eumartii* Carpenter, *F. radicicola* Wollenw., *F. oxysporum* Schl., *F. sulphureum* Schl., and *F. coeruleum* (Lib.) Sacc.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Although Fusarium rots occur in a wide range of truck-crop plants and are an important factor, especially in storage and during marketing, serious study has been made only of those that affect the potato and the sweetpotato. Generally Fusarium rots are most common in the tubers. When these fungi attack other parts of growing plants the symptoms complexes have received other descriptive names such as foot or root rot, wilt, or yellows. In most cases the parasite has not even been isolated or identified, and naturally little exact knowledge is available concerning the relation of environmental factors to the rots. This discussion of the rots of the potato, while not strictly applicable to those of other hosts, covers the general principles.

Unlike the Fusarium wilts, Fusarium tuber rots of the potato occur in potatoes from all parts of the United States. With the exception of stem-end rot (*F. eumartii*) and black field rot (*F. radicicola*), they do not occur in the growing tuber in the field. They are destructive in tubers primarily during storage and transit,

and the losses caused by them probably are exceeded only by those due to bacterial soft rot.

Some of the species that cause tuber rot also attack the growing plant, destroying the root parenchyma or invading the vascular elements or both, and cause blight or wilting. (See Fusarium Wilts, p. 25.)

Although the symptoms depend in part upon the species of the parasite and upon external conditions, especially temperature and moisture, a fairly general characterization of the rots can be given. The disease generally is marked by sunken, shriveled, wrinkled, or broken areas occurring on the tuber surface at the stem end (*Fusarium radicicola*, *F. eumartii*, and *F. trichothecioides*), at the eyes, at the lenticels, or anywhere between them. (Pl. 8, A, B, D; pl. 9, A.) As a rule these areas are brown to black, and on them may appear masses of whitish, dark, or brightly colored mold. The tissue underlying such discolored and sunken areas varies in discoloration and consistency, depending upon the species causing the rot and upon the conditions under which the rot develops. The rot may be wet and jellylike (jelly end rot, *F. radicicola*, pl. 9, C), even mushy and leaky (*F. oxysporum*, pl. 7; *F. radicicola*, pl. 8, A), or dry and brittle, and with or without cavities lined with whitish or brightly colored, fluffy or powdery molds (pl. 8, C). The color of the affected tissues varies from light to dark brown and even black. Sometimes several species attack the same tuber, causing a combination of symptoms. Fusarium tuber rot is usually dry at low temperatures and wet at high. It never is slimy, even when wet, and never has a bad odor unless accompanied by other fungi or bacteria. In some types of the rot, as in powdery dry rot (pl. 8, C) for example, the fungi have a tendency to rot the pith and inner phloem parenchyma faster than the tissues outside the vascular ring, so that a spot which is scarcely apparent on the surface may be underlain by an extensive wet region or by a rotted, dry interior, full of cavities and surrounded by a shell of healthy cortical tissues. (Pl. 7, B.) In other types the rot progresses more evenly into the tuber, destroying the various tissues at more or less equal rates. In some types the dried-out stage is often marked by layerlike, dry, tough remnants of tissues. In some types there is merely vascular discoloration (*Fusarium oxysporum*, pl. 9, D; *F. eumartii*); in others there is discoloration of the vascular tissues in advance of the rot, appearing as a network or flecks; and finally there is stem-end rot with vascular discoloration, accompanied by a glassy appearing brown rot of the adjacent tissues, which often extends throughout the length of the tuber (*F. eumartii*, pl. 9, E).

Unlike many diseases that are caused by generalized parasites and do not have lesions with diagnostic characters, but in which the parasites themselves are characteristic signs, the various Fusarium rots of potatoes and other crops can not always be identified by the structures of the parasite. Superficial mycelium does not occur invariably; when it does, it may occur as a cottony mass, which may remain white or may take on distinctive bright colors, or it may break through the surface in little tufts of white or brightly colored molds (pl. 8, A, B, D), as in some potato and onion rots. Neither the mycelium nor the spores are ever black.

Their bright color is the most distinguishing macroscopic character, white, orange, and ocher (pl. 8, A, B, C) being some of the colors found in conidia, while the mycelium may be carmine, blue, or brown. Not all Fusaria produce sclerotia. These are rarely produced on host tissues, and when they occur they are not distinctive. There are no macroscopic reproductive structures like the sporangia in Rhizopus. In fact, one must resort to the microscope to make a diagnosis even when a brightly colored mycelium is involved. This does not always decide the matter, for the characteristic sickle-shaped, dorsiventral, septate conidia which frequently give color to the mycelium often are absent. Microconidia may occur but usually are not characteristic enough for positive identification.

CAUSAL FACTORS

Like other Fusarium species, those parasitic on potato tubers are inhabitants of the soil, where they live saprophytically on plant débris. Although it appears that no soils are entirely free from pathogenic Fusaria, various soils and localities seem to be characterized by either the presence or the predominance of certain Fusaria. In general, the Fusaria that induce wilts have high temperature optima. Consequently, the Fusarium wilts are limited to or predominate in sections or seasons marked by hot weather.

In the rots the type varies with different sections, being determined by the species of Fusarium that either characterize the particular section or happen to predominate during the growing season. Thus the Fusarium rot that usually occurs in the Eastern States is induced by *F. coeruleum;* the rot in the Southern States is induced by *F. oxysporum* and by *F. radicicola* (pl. 8, A; pl. 9, A, C), which also induces black field rot in Idaho and contributes to jelly end rot of the Burbank variety in the irrigated sections of the far West. (Pl. 9, C.) The powdery dry rot in the middle western plains and Mountain States east of the Cascade Mountains is induced by *F. trichothecioides*, which also occasionally induces wilt (pl. 8, C, D), while the rot in potatoes west of the Cascades is induced by *F. coeruleum.*

As pointed out before, four species are known to invade the tuber or plant principally in the field. *Fusarium radicicola* apparently can invade tubers through the lenticels. *F. oxysporum*, *F. eumartii*, and to a minor degree *F. trichothecioides* can invade the growing plant and from it the young tubers.

All the other parasites are wound parasites and consequently become important after harvesting. The rough handling of tubers after harvest naturally provides many points of entry. Infection proceeds from the mycelium by contact or from spores carried on the tubers, in the soil, on containers and implements, and in storage houses.

Whether or not infection will take place and the length of the incubation period are determined primarily by moisture and temperature conditions. The rate of formation of wound periderm and of suberization is most important in determining infection. At a storage temperature of 44° F. these form so slowly that they offer very little resistance to the growth of the Fusaria. In the Rural New Yorker variety it requires 3 or 4 days at 70°, or more than 10

days at temperatures below 50°, for the formation of wound periderm that is effective in excluding *F. coeruleum* and *F. sulphureum*. The various species of Fusarium have different temperature requirements. *F. oxysporum* and *F. eumartii*, which can also induce wilt, do not grow at all at temperatures below 50°, grow best at 86° and 78°, and cease growth at 100° and 95°, respectively. *F. radicicola*, which induces black field rot and also jelly end rot, does not grow at temperatures below 53°, grows best at 86°, and ceases growth at 100°. *F. trichothecioides*, which induces dry rot and some wilt in the plains and Western States, does not grow at temperatures below 41° or above 86°, and grows best at 78°, at which temperature it also induces most rapid rot. *F. sulphureum* and *F. coeruleum* are able to infect tubers and cause a progressive decay at the lowest temperatures feasible for storing potatoes.

Under most favorable conditions the incubation period is 4 to 5 days. In 10 to 15 days as much as one-half to three-fourths of medium-sized tubers may be destroyed. At 77° F. tubers frequently are completely destroyed in two to seven weeks. At low temperatures (50° or below) most Fusaria grow only slowly, but the growth made at these temperatures may be sufficient to start infection and to establish the organism so that it is ready to make very rapid growth as soon as it is exposed to higher temperatures. Most of the rot-producing Fusaria that have been studied can endure temperatures below 41° or even freezing and remain viable for long periods.

CONTROL MEASURES

Control of the Fusarium diseases is not an easy matter. In the first place, the wilt diseases and the decays require different treatments. Rotation of crops has proved beneficial with some of the wilts, but in general it has not been as helpful as in other diseases because of the saprophytic habit of the organisms and their ability to feed on a wide range of hosts. The selection and development by breeding of resistant strains or varieties has proved a much more promising method of control with a number of other crops.

Although all soils, even virgin soils, seem to be infested with several species of Fusarium, it is well known that pathogenic species have either been introduced or made economic factors by cultural practices. Failure to rotate crops frequently makes the Fusarium problem acute. The light soils of the South that are suitable for watermelon culture usually will produce only one crop of watermelons free from wilt. It is well known that the powdery dry rot of potatoes is a far more serious storage rot in cellars in which rotting tubers were stored or allowed to accumulate than in new ones or in those which are kept clean and free of débris and in which a gaseous or liquid disinfectant is used.

The safest and surest methods of controlling the rots that are induced by wound parasites are the careful handling of tubers to avoid cutting and bruising, the sorting out of bruised, broken, cut, diseased, or frozen potatoes, and storing the tubers in well-ventilated places at temperatures between 36° and 40° F. Wound infection and rot can not be prevented in storage potatoes by manipulation of moisture and temperature conditions alone. (*25*, *49*, *87*, *90*, *111*, *137*, *168*, *186*, *190*, *191*.)

FUSARIUM WILTS

(*Fusarium oxysporum* Schl.; *F. eumartii* Carpenter; *F. trichothecioides* Wr.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Fusarium wilts occur in the warmer potato sections of the country or during warm seasons. Fusarium wilts of the potato not only reduce the yield by killing plants prematurely but also cause discoloration and decay so that tubers become unmarketable. In addition they open the way for aggressive tuber-destroying fungi. Discoloration in the vascular bundles without decay counts heavily against seed stock.

In other plants, Fusarium wilts generally are important because affected plants, if not killed outright, are discolored, or when they are free from lesions, are small, flabby, or ill-shaped, and thus have a lowered market value.

In the tubers the disease is marked by mere brown discoloration of the vascular ring (pl. 9, D, E), or it may be a stem-end rot of the tuber (pl. 9, A. C, E). In the case of stem-end rot (*Fusarium eumartii*) the sunken areas at the stem end occur with wrinkling around the margins. (Pl. 9, A.) When there is only discoloration, cutting is necessary to make it visible. In the field as much as a quarter of the tuber may became involved from the stem end. The rot displays a variety of symptoms: (1) Stem-end rot with no vascular discoloration and a sharp line of demarcation between the healthy and infected tissues (pl. 9, A, B); and (2) stem-end rot with vascular discoloration which may be associated with a soft rot of the adjacent tissues and may extend the entire length of the tuber as a band of light-brown tissue. (Pl. 9, D, E.) (See Fusarium Tuber Rots, p. 21.)

In the growing plant *Fusarium oxysporum* and *F. eumartii* induce somewhat different symptoms. The wilt induced by *F. oxysporum* is marked by yellowing around the margins of the lower leaves, which gradually develops in the entire top, accompanied by complete wilting. This yellowing and wilting usually affects one side of the plant more severely than the other. In the wilt produced by *F. eumartii* the first symptoms are light-green mottling of the leaves accompanied by the irregular bronzing of the upper surface. Under dry conditions the leaves slowly turn yellow; in severe cases there is leaf dropping, with the leaves hanging to the stem by a mere thread. Sometimes an internal blackening of the veins, which may be visible through the tissues, develops in the leaf blade and petiole. Under dry conditions the wilt progresses rapidly, and the entire plant wilts and dies in a short time. If there is an abundance of moisture, wilting does not result, but there is a yellowing, rolling, and rosetting of the top, which may be accompanied by the development of aerial tubers. In both types the fine roots are destroyed. Under wet conditions the cortex sloughs off, and under dry conditions the roots become brittle. In the former case there is discoloration of the vascular tissues of the stem, which, however, does not extend above the ground. In the latter there is brown discoloration of the vascular bundles, which is accompanied by necrosis of the inner and outer phloëms, and the discoloration ex-

tends into the upper stem and foliage, often being very pronounced in the pith at the nodes.

Frequently, wilts induced by Fusaria can be distinguished from those induced by bacteria by the absence of bacterial ooze in the affected tissues, but very often they can not be distinguished save by cultural methods. Verticillium wilt of the potato and other plants can generally be distinguished from Fusarium wilt only by laboratory determination of the parasites. Verticillium wilt of the potato occurs north of the range of Fusarium wilt. (See Verticillium Wilt, p. 50.)

CAUSAL FACTORS

Potato wilt is generally induced by two species of Fusarium, *F. oxysporum* and *F. eumartii*, and occasionally by *F. trichothecioides*, which, like other Fusarium species, are inhabitants of the soil, where they live saprophytically on plant débris and from which they may attack the plant. In the potato the parasites may attack the plant either from the soil where they overwinter or from infested seed pieces. In the case of *F. oxysporum*, most of the infection apparently proceeds from the soil into the roots. In the other types wilt is reproduced much more frequently in plants arising from infected seed. Nevertheless, infection from the soil through the roots is generally more severe and consistent in the latter type. In both cases the fungus grows from the mother plant through the stolons into the new tubers. There is no spread of the disease in transit or storage from tuber to tuber. The killed tissues, however, are subject to invasion by secondary organisms.

The temperature requirements of *Fusarium oxysporum* and *F. eumartii* are somewhat different. The former requires at least 57° F., with heavy soil inoculation, to produce the disease. The most rapid development of the wilt takes place at temperatures above 86° and up to the growth limit of the fungus, which is about 95°. Infection is favored by high soil moisture, but after infection has set in development of the wilt is faster in dry soil. The lowest soil temperature at which *F. eumartii* can produce wilt is 52°, and the highest is 86°. The most favorable temperature for infection and development of the rot is from 68° to 77°. Infection occurs in wet and dry soils, taking place more readily in wet soil, but the wilt subsequently develops more rapidly in dry soil. Tuber infection takes place more rapidly in wet soil.

The symptoms of wilts in other plants are essentially those found in the potato wilts. In these, as in the potato, blighting or wilting, often accompanied by prompt or delayed death, may be the result of destruction of the roots or foot of the plant, of general or local plugging of the water vessels (tracheomycosis), or the killing effect of the toxic substances liberated by the parasite or by the interaction of host and parasite, or a combination of these. Some of the wilt producers are also parenchyma destroyers or rot producers.

Wilt may appear at any time in the season, and once it has appeared, death of the foliage may occur slowly and fitfully or very rapidly and suddenly. The disease may appear so early, especially when it proceeds from potato seed pieces or sweetpotato roots, that the sprouts are killed before they emerge from the ground. Fre-

quently it appears in young plants, which may die prematurely or live to maturity. Finally, it may not appear until a short time before the crop is made.

CONTROL MEASURES

Since infection may proceed from the soil or from infected seed potatoes, two lines of control must be followed for potato wilt. If it is known that a given tract of land has produced a wilt-affected crop of potatoes, a long rotation should be used before potatoes are planted there again. There is some possibility that virgin lands may be infested with the organisms. Fields on which crop rotation has been practiced should be used as far as possible. Infected seed should be discarded. Since the parasite in a tuber is not necessarily visible, and since not all tubers with vascular discoloration are infested with Fusaria, the surest way to get clean seed is to practice roguing in the seed plot or to purchase only certified seed. Since tubers get the disease from the mother plant, it frequently is possible to prevent invasion of the tubers by the parasite, by digging a week or two early if the disease has not yet extended into them. (*33*, *34*, *51*, *62*, *87*, *97*, *99*.)

(For additional control measures, see Fusarium Tuber Rots, p. 21.)

GREENING (SUNBURN)

Greening of the tissues of the potato tuber results from exposure to light, either in the field or after harvesting. It is more commonly found in immature than in fully matured tubers. Affected tubers are discriminated against in the market, because they are bitter and unfit for food.

Ordinarily, the green color is restricted to the layers of cells immediately below the skin. If exposure to the light is prolonged, however, chlorophyll develops in the deeper tissues. (Pl. 5, F.) Once the bitter taste accompanying the green color has developed, it does not disappear completely even though the greened tubers are removed from the light. Often the affected tissues become tough, leathery, and excessively yellow, without developing chlorophyll. (*176*, *p. 32*.)

GROWTH CRACKS, KNOBBINESS, AND SECOND GROWTH

It is generally assumed that deep cracks (pl. 10, B) on the surface of tubers, knobs (pl. 10, D), pointed ends (pl. 3, D), and second growth are the result of uneven or unfavorable growing conditions. Although it is true that these malformations may appear on all varieties in all sections of the country under unfavorable conditions of growth, it is also a general observation that these deformities are produced under identical soil and climatic conditions on the tubers of some plants, whereas those of other plants are free from them. It has been contended that the latter situation is due to the fact that these deformities are often associated with abnormal vine symptoms. Abnormally elongated tubers (pl. 3, D) especially are associated with a condition of the tops that is believed to be due to a virus. The top symptom is known as marginal leaf roll, while the tuber symptom is called spindle tuber. (See Spindle Tuber, p. 49.) Knobbi-

ness and cracking of tubers and an internal browning or spotting are reported as being associated with a vine condition known as yellow dwarf. Further experimental work will have to determine whether these observations and interpretations are correct.

It has been established experimentally that these deformities can be induced by an uneven water supply and by indiscreet use of fertilizers. The symptoms indicate checking of growth, followed by renewal with resultant cracking of the hardened skin or development of knobs at the buds, or elongation. Growth cracks usually are covered with the normal cork layer and therefore are not subject to decay. The knobs and pointed ends, however, especially when they are due to recent excess of water, are immature and tender. Occasionally they contract decay in the soil. (See Jelly End Rots, p. 29.) The knobs are easily broken off, and an easy entrance for rot-producing fungi is thus afforded. In the long Burbank and the Netted Gem varieties in the irrigated sections of Idaho and California second-growth knobs are a common occurrence, and the wounds made when they are accidentally or deliberately broken off open the way for leak and Rhizopus rot. (See Leak, p. 33, and Rhizopus Soft Rots, p. 40.)

In so far as these abnormalities are due to climatic conditions, little can be done to control them. The maintenance of proper soil moisture by frequent cultivation during drought periods is recommended. In the irrigated sections a great deal can be done to prevent second growth by proper handling of the water supply. Care should be taken to avoid breaking off the knobs. (*12; 167, p. 35–36.*)

HOLLOW HEART

Hollow heart is a condition brought about by a too rapid or an irregular rate of growth. It is common in certain large-tubered rapid-growing varieties during wet seasons or in very fertile soils, and is more likely to occur in large than in small tubers. Largeness of tubers, however, is not an infallible sign, because small tubers also may be affected. There is no absolutely reliable external symptom. Hollow heart seriously affects the marketability of potatoes.

Hollow heart consists of more or less irregular cavities of varying size in the center of the tuber. (Pl. 10, A.) The cavity is usually lined by dead tissue that is glassy and brown. Hollow heart can be distinguished from black-heart (see Black-heart, p. 10) in which cavities have developed by the conspicuous discolorations associated with the latter. Hollow heart does not develop in transit or in storage. So far as known, affected tubers are not unsuited for seed purposes.

Control measures involve regulation of the rate of growth of the developing tubers. Seasonal and climatic conditions determine to what extent the grower can force development of his crop by use of fertilizers or by irrigation. His experience will also guide him in selecting varieties that do not grow with abnormal rapidity.

IMMATURITY

Maturity is a very important factor in determining the market quality of tubers. Lack of maturity, or immaturity, is indicated

by several characteristics. Mature tubers have a well-developed layer of corky tissue that protects them from excessive drying out, bruising and peeling in handling, and rapid greening in light. Immature tubers can be recognized by excessive wilting and flabbiness (pl. 10, D), by excessive peeling or feathering of the skin, and by the readiness with which they turn green. A considerable amount of this is tolerated in the southern crop and in the early crop of the North, which, however, are marked for immediate consumption. The immature stock is very susceptible to invasion by numerous rot-producing fungi, especially if it is shipped wet and dirty in overheated cars or in tight barrels.

Immature stock should be handled as carefully as possible, if bruising (pl. 10, C), with subsequent drying out and decay, is to be avoided. To avoid loss of water through the unbroken skin, the tubers should be protected from excessively dry air. Greening can be prevented by keeping them out of direct sunlight. The safest way, however, to prevent the ills attendant upon immaturity is to allow the tubers to grow until they develop a normal layer of cork.

INTERNAL BROWN SPOT

The causal complex of internal brown spot has not been determined. The development of this disease in many cases seems to be associated with dry soils or lack of water at some period during growth of the tuber. It seems to be more common and serious in some varieties than in others. It affects the marketability once its presence in a lot is detected. There are no external symptoms, and consequently affected tubers often reach the ultimate consumer before complaint is made.

Internal brown spot is marked by brown or yellow regions of dead tissues scattered throughout the tuber. The affected areas may be small or large, even one-half inch in diameter. (Pl. 6, C.) They may be scarce or numerous, and they are isolated, and not connected as in net necrosis. The color of the lesions is very much like that described under Drought and Heat Necrosis (p. 16). (Pl. 6, D.) Possibly the essential difference is merely one of the extent of the tissues affected.

There are no experimental data available on the basis of which any definite statement as to cause, relation of environmental factors, and control can be made. It is a moot question whether bacteria or fungi are directly involved in the production of the necrotic areas. It is advisable on general principles not to use affected tubers as seed stock. There is no evidence that the disease develops or becomes more serious in transit or in storage. (*9; 12; 167, p. 29.*)

JELLY END ROTS

(*Rhizoctonia solani* Kühn[12] and various Fusarium species)

OCCURRENCE, SYMPTOMS, AND EFFECTS

At times the blackleg organism (see Blackleg, p. 11) produces symptoms that are indistinguishable from jelly end rot, although it appears now that most jelly end rot is induced by *Rhizoctonia*

[12] Syn., *Corticium vagum* Berk. and Curt.

solani. This disease occurs in the Burbank variety in California and in the Netted Gem in Idaho. Its economic significance lies in the fact that it lowers the market value of potatoes or renders them entirely worthless and also opens the way for secondary rot-producing organisms.

The decay usually occurs in those portions of the tuber which are succulent and deficient in starch, such as the pointed ends of the Burbank type and occasionally also in the knobs and at the eye end. (Pl. 9, C.) Before drying out, the affected tissues have a clear, jellylike consistency. Later on, they shrivel down almost to the healthy flesh underneath. The decay develops little or not at all in transit or storage, since affected portions usually dry out immediately after digging.

CAUSAL FACTORS

The fungus *Rhizoctonia solani*, which induces most cases of jelly end rot, overwinters as black scurf on tubers or in the soil, from where it may infect growing tubers. The disease does not develop or spread in transit or in storage, although it opens the way for Fusarium species and bacteria.

(For a detailed discussion of the relation of environmental factors, see Rhizoctonia Black Scurf or Soil Rot, p. 37.)

CONTROL MEASURES

Prevention of jelly end rot depends on the ability of the grower to provide cultural conditions that will prevent the development of pointed tubers. This, in turn, depends upon the proper handling of irrigation water. Tubers that show active stem-end rot in the spring should not be used for seed, because they harbor secondary organisms that can cause losses. (*164*.) (See also Spindle Tuber, p. 49.)

LATE BLIGHT (TUBER ROT)

(*Phytophthora infestans* (Mont.) DBy.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Late blight occurs in all varieties of the potato in varying degree. It affects the leaves, stems, and tubers, causing such heavy losses in the sections best suited for potato culture that it has come to be known as the potato disease.

From the point of view of production, the losses are due to poor germination of affected seed pieces, to destruction of the young sprouts and the green food-making parts of the plant so that poor yields result, and to attack upon tubers so that these either are unmarketable or are marketable only at a reduced price. Losses are not restricted to the field, however, as the disease destroys tubers in transit and in storage. The disease may cause serious damage in any potato-growing section where diseased tubers were planted, whenever very moist, cool weather prevails during the early growing season, followed by moist, moderately warm weather later in the summer. It occurs frequently in severe form in the New England States adjacent to Canada, in the Puget Sound district, and less fre-

quently though often in severe form in New York, New Jersey, Pennsylvania, Ohio, Michigan, Wisconsin, Minnesota, Iowa, in the mountain districts of the Eastern and Western States, and in Florida on the early crops.

In the earliest stages the disease on the tubers is marked by reddish-brown, brown, or purplish-brown spots (pl. 11, D, E, F), which, as development continues, become shrunken and darker colored. In storage stock late-blight tuber rot is moderately dry, firm, or leathery, and does not usually extend more than one-eighth to one-fourth of an inch beneath the skin (pl. 11, A, B); but in newly harvested potatoes, particularly those of the early southern crop, the reddish-brown moist decay often penetrates to a depth of half an inch or more. (Pl. 11, C, F.) The decay may also occur in the tissues at the stem end as a broad brown ring, and in advanced stages the tuber may be converted into a shriveled mass. In newly harvested stock or in stock held in very moist conditions, the white tufts of the fungus often appear at breaks in the skin and at the lenticels.

A serious feature of the disease on tubers is that it opens the way for bacteria and other fungi which rot the tuber much more rapidly than the late-blight fungus. If bacteria get in through diseased spots, a very wet, soft, or slimy and foul-smelling rot results. (See Bacterial Soft Rots, p. 6.) This usually occurs under warm, moist conditions. If other fungi (usually Fusaria) follow it, an odorless rot results which may be wet or dry, depending upon temperature and moisture conditions. (See Fusarium Tuber Rots, p. 21.)

Late blight on the growing plants reduces the yield and under favorable weather conditions may cause the complete loss of entire fields. In the earliest stages it is marked by water-soaked, pale greenish brown spots on the leaves and stems. The spots often begin at the margins of the leaves and eventually cause them to wilt and blacken. In dry weather they shrivel and dry up. When the weather is warm and moist the spots enlarge rapidly, and one spot may kill an entire leaf in one to four days. Under such conditions a white coating of the fungus grows on the lower or upper sides of affected leaves and on the stems, bearing spores in great numbers.

CAUSAL FACTORS

The fungus *Phytophthora infestans*, which induces late blight, overwinters in tubers in storage or in the field. It is not known to overwinter in the soil, although the behavior of the disease at times indicates this as a possibility. Experiments have shown that the organism can spread to the soil and remain parasitic and pathogenic for at least 57 days.

The fungus grows into sprouts from diseased tubers and in this way is carried to the surface of the ground, unless the sprouts are killed before they break through the soil. The fungus may spread in or on the soil for an inch or so if the ground is very wet, or it may grow on the diseased sprout during wet weather and produce great numbers of spores. These may be carried by wind, rain, or insects such as the potato beetle and slugs to near-by healthy plants and start new disease spots. From diseased vines the spores either

fall or are washed to and into the ground by rains, and consequently tubers which are borne within an inch or two of the surface or which are not completely covered with soil become covered with the spores of the fungus. In wet seasons the fungus grows so well in heavy soils and produces its spores so freely on and in the soil that all tubers in the ground may become thoroughly covered with them. The fungus may also grow into the stem end of tubers from a diseased mother plant or from the soil, entering by way of the stolon, but this rarely happens.

Tubers also become thoroughly covered with spores if they are dug before frost while the disease is active in the field or if they are piled in the field and covered with diseased vines. When conditions are favorable for the fungus in transit and storage, spore production and infection may take place there also.

The fungus does not grow if moisture is not abundant or if the temperature is below 40° or above 86° F. It grows best at temperatures between 73° and 80° and when the air or soil or tubers are very moist. Under these conditions it sporulates most abundantly. The spores do not germinate if they are dry or if the temperature is either below 36° or above 86°. They germinate best at about 53°, either in a film of water or in very moist air. Exposure to dry air for six hours kills them. According to laboratory studies conducted with leaves, 50° to 55° are the most favorable temperatures for infection. The optimum temperature for tuber infection has not been determined, but it probably does not vary much from that of leaf infection. Three to twenty-four hours are required for leaf penetration under the most favorable conditions; the period necessary for tuber penetration is not known. It probably is at least as long as the former. Symptoms of the disease do not appear on the leaves for four or five days after the fungus comes in contact with them. In moist air with a temperature between 60° and 75° the disease progresses so rapidly that it may kill the entire plant in one to four days.

The presence of the fungus on a tuber does not necessarily mean that the tuber will become infected. Temperatures below 40° or above 86° F. or dryness will prevent development of the inoculum. On the other hand, since some time must elapse between penetration and the appearance of the first symptoms, it frequently happens that tubers are stored or loaded that look sound but in which the fungus has established itself and which will develop symptoms only if temperature and moisture conditions are favorable. The fungus may lie dormant in tubers for months and then begin to grow as soon as favorable temperature conditions arise. In some experiments it was found that inoculated tubers did not develop the first symptoms for three weeks at 64° to 71° in moist air. Apparently sound tubers taken from a field where the tops were killed by the fungus were stored at 40°, 55°, and 70°, and showed 17, 53, and 19 per cent, respectively, of rotting tubers at the end of two months. In moist air with temperatures above 41° the fungus grows out of diseased tubers and produces spores that can infect near-by tubers with unbroken skin. It has been found that in storage conditions, with moisture and temperatures between 60° and 71°, lesions on tubers may enlarge at the rate of 2 inches in 10 days.

CONTROL MEASURES

Diseased tubers should not be used for planting, since they carry the parasite to the field.

Spraying with Bordeaux mixture[13] will kill the fungus and its spores on the surface of the leaves and therefore control the disease in the field if it is done systematically, thoroughly, and early enough, with spray machinery that gives 150 pounds pressure or more. Whether to spray and when to spray depend upon the locality and the weather. In localities where blight usually occurs, spraying should be begun when the plants are 6 inches high and be repeated at intervals of a week or 10 days, or frequently enough to cover the new foliage as it appears.

It is not generally advisable to dig a diseased field while the soil is wet. If potatoes grown on light soil become badly affected and the weather turns dry and frosting time is near, it is advisable to delay digging until after a frost, which kills both the fungus and the vines.

If a crop grown on a very wet, heavy soil becomes diseased during wet weather, it may be advisable to dig at once in order to save some of the crop, even though the chances of losing much of it are very great.

Piles of tubers in the field should not be covered with diseased vines, as the disease may thus be carried from the vines to the tubers. If tubers in transit or in storage are kept dry and at temperatures between 35° and 40° F., the disease will develop little, if any. (*22*, *42*, *74*, *91*, *93*, *103*, *108*, *116*, *118*, *120*, *121*, *144*.)

LEAK

(*Pythium debaryanum* Hesse, and other Pythium species)

OCCURRENCE, SYMPTOMS, AND EFFECTS

The Pythium organisms attack not only the potato but also induce a soft watery disintegration, known as leak, of fruits of cucumber, pepper, muskmelon, and watermelon. For the potato, the disease was once considered characteristic of tubers grown in the delta lands of the San Joaquin Valley of California. It now appears to be virtually coextensive with the potato crop of the United States, having been noted in the markets in potatoes from all sections.

The occurrence, symptoms, and effects of leak are practically identical with those of a decay of tubers induced by Rhizopus species (see Rhizopus Soft Rots, p. 40) and very similar to those of pink rot (see Pink Rot, p. 35). A positive separation of the first two rots is not possible without microscopic or cultural studies.

Although Pythium species are important field parasites for a great number of truck crops, producing a soft, cortical decay of the underground parts of the stem and of roots, especially in seedlings under crowded seed-bed conditions, they seem to be of economic significance in potato culture only through their attacks upon the tubers during and immediately after harvest.

The most characteristic symptom of leak is the extremely watery nature of the affected tissues. (Pl. 12, C. D.) The water is usually held by the disintegrated tissues, but when pressure is applied a

[13] Copper sulphate (bluestone) 5 pounds, stone lime 5 pounds, water 50 gallons.

yellowish to brown liquid is given off readily. Another characteristic is the granular nature of the diseased tissues. (Pl. 12, D.) Externally the affected tissues appear turgid and may show discoloration ranging from a metallic gray in red varieties to brown shades in white and dark skinned varieties. (Pl. 12, C.) Internally the affected tissues are creamy in color to begin with. Upon cutting, as well as in the later stages of the disease, they soon turn tan or slightly reddish and then become brown and finally inky black. The diseased areas generally are not sharply set off from the healthy tissues. (Pl. 12, D.) There is rarely any visible fungous growth externally or internally, nor are there cavities lined by white or brightly colored molds, as in the soft types of Fusarium rot, which somewhat resemble leak. The colors of black-heart resemble those of leak very much, and frequently these diseases occur simultaneously. Tissues affected with black-heart, however, are not soft, watery, and granular, as are those showing leak.

CAUSAL FACTORS

Pythium species as parasites are among the most common and serious soil fungi and are responsible for a great deal of damping off in seedlings and for various root rots.[14] Infection in the field therefore proceeds from the soil. In the potato, infection by *Pythium* spp. apparently takes place only through breaks in the skin of the tubers. It frequently occurs in tubers that are affected with sunburn or sunscald, especially when they are allowed to lie in or on hot soils after they are dug. Infection can also take place in transit and storage by contact.

Hot weather and abundant moisture seem to favor infection and development of the disease. In those potato-growing sections in which the crop is harvested and moved during extremely warm weather (such as the early Idaho and Washington crop and the crop grown in the San Joaquin delta of California), leak is a constant and serious factor. In other sections it seems to occur only if there is abnormally warm weather during the digging, moving, and early storage of the crop. When infection has once taken place the disease progresses very rapidly and develops in inoculated tubers even in refrigerator cars. Leak may develop in transit or in storage in tubers which show no visible breaks of the skin and which, when taken out of the field, appear to be free from the disease. At temperatures between 60° and 90° F. the disease progresses so rapidly that lesions become visible within 36 hours after inoculation. Freezing temperatures arrest the development of the disease, so that the brown to inky black lesions frequently dry out, forming cavities. At low temperatures (32° to 50°) leak is frequently followed by Fusarium tuber rot. Under warmer conditions bacteria invariably get in and arrest the growth of Pythium, so that its presence is difficult to demonstrate. The typical symptoms of bacterial soft rot develop when bacteria get in.

CONTROL MEASURES

Control of leak involves keeping tubers as cool and dry as possible during harvest and loading and in the early stages of transit and

[14] *Rhizoctonia* sp., *Fusarium* sp., *Aphanomyces* sp., and other fungi also induce damping off, seedling blight, and root and stem rots.

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A, B. Scald, caused by exposure to sun after digging. A, local type; B, general type

C, D. Leak (Pythium species)

The lesions in Rhizopus soft rot and in pink rot are very similar to these

storage and in avoiding breaks in the skin. (*10, p. 234–247; 23, 63, 88, 180.*)

NET NECROSIS

OCCURRENCE, SYMPTOMS, AND EFFECTS

The causal complex of net necrosis has not been established experimentally. It is possible that various factors, both parasitic and nonparasitic, may produce symptoms which are now included in the term "net necrosis." Plants affected with leaf roll usually produce some tubers showing a net necrosis, and such tubers generally give rise to plants affected with leaf roll. However, not all tubers produced by a plant affected with leaf roll show net necrosis.

The outstanding characteristic of net necrosis is the presence of a network of necrotic strands, brown to brownish black in color and sometimes extending throughout the flesh of the tuber. (Pl. 6, E.) In mild cases the netting may be limited to a small portion of the tuber, usually at the stem end. In typical cases it is never confined to the vascular ring.

In view of the association of tubers showing net necrosis with plants affected with leaf roll, it will be necessary to describe the symptoms of leaf roll. Affected plants show an upward rolling on the midrib, which gives the leaves a tubular shape and the plant a stiff, erect appearance. A rolling of the lowermost leaves, accompanied by leatheriness or brittleness, is the most constant diagnostic symptom. The lower leaves often die back from the tips. Frequently the affected plants are bushy and even rosetted and are almost always paler than normal ones. The yields are reduced very considerably. A characteristic symptom is necrosis of the phloem elements of the plant.

CAUSAL FACTORS

Experiments have demonstrated that the causal agent can be transferred to healthy plants in the juice of diseased plants by mechanical means, but there is no unanimity of opinion as to the nature of this causal agent.

No data are available concerning the relation of the disease to environmental factors. It is known that the symptoms are most pronounced when conditions are most favorable for growth of the potato plant, and that unfavorable conditions lead to no development or imperfect development of symptoms of the disease.

CONTROL MEASURES

The only effective control measure known at present is the roguing of seed plots and the use of certified seed obtained from such plots. (*43, 158; 167, p. 30.*)

PINK ROT

(*Phytophthora erythroseptica* Pethyb. and *Phytophthora* sp.)

There has been noted occasionally in the United States a decay of tubers which apparently is identical with the pink rot of European potatoes. The rot has been found in only a few instances in stock from Idaho, Kentucky, and Oklahoma. The fungus *Phytophthora*

erythroseptica has been demonstrated to be a factor in the causal complex of European pink rot, but from American tubers with apparently identical symptoms an undescribed Phytophthora species has been isolated.

It has been demonstrated that *Phytophthora erythroseptica* lives in the soil in Europe for at least four years and remains potentially parasitic and pathogenic for both vines and tubers during this period. A wilt and stem decay sometimes is part of the symptom complex, but generally rot of tubers is the only or the most important expression of the disease. Decay usually begins at the stem end and may progress rapidly throughout the length of the tuber. The affected tissues are firm and resilient but under pressure exude water freely like those in leak. (Pl. 12, C, D.) The affected tissues are almost normal in color until they are exposed to the air, when the flesh turns a characteristic pink. After several hours' exposure the color gradually darkens and finally becomes purplish brown to black. (See Black-heart, p. 10.) Externally the decayed tubers have a dull brownish or black and dead appearance, and the lenticels stand out as black spots. Resting spores of the fungus live over winter in and on decayed tubers and stems, thus contaminating the soil and making it unsuitable for future potato culture.

The most feasible control measures are crop rotation and the practice of sanitation such as the burning of diseased vines and tubers. (*27*, *32*, *134*, *135*.)

POWDERY SCAB

(*Spongospora subterranea* (Wallr.) Lagerh.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

When powdery scab first made its appearance in the United States dire results were anticipated. It has since developed that the climatic conditions in most of the potato sections of the country are such that the fungus does not grow well, and consequently the disease is not of much importance. Its economic importance, to some extent, lies in the fact that the underground parts of the growing plant may be attacked, with resultant decrease in yield, but more in the fact that the tubers are disfigured, so that their market value is decreased or destroyed. The disease occurs only in the cool potato sections, such as those of the North, especially in the Northeast. It also occurs in the cool sections of the Pacific Northwest. Its severity depends upon climatic conditions.

Although this disease is known as a scab, its appearance is quite unlike that of common scab. The individual spots are more nearly circular and on the average smaller than the lesions of common scab. When fully mature, they are open pustules filled with a brown, more or less powdery mass of spores and broken-down tissue, surrounded on the edge by fringed remnants of the tuber skin. (Pl. 13, C. D.) In the immature stage these spots are closed and somewhat raised, and dark on the outside. Under favorable moisture and temperature conditions similar lesions develop on the roots and stem.

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A, B. Wart, a serious disease. Suspicious cases should be reported promptly to county agent or agricultural college

C, D. Powdery scab

CAUSAL FACTORS

The slime mold, *Spongospora subterranea*, which induces the disease, lives over in the soil, from which infection proceeds. The organism also may live over in stored tubers and be carried on these into clean soil. There is no spread of the disease from tuber to tuber in transit or in storage. Observation indicates that powdery scab is favored by wet soil and cool weather. Experiments have shown that the disease does not develop if the soil temperature is much above 60° F., nor if the soil is dry. For development the soil must be moist; the more moist the soil, the more severe the disease. Under favorable moisture and temperature conditions the lesions on tubers may enlarge in storage. The constant irritation of the tissues with but slight cork development results in excessive drying out and opens the way to invasion by secondary fungi, such as various species of Fusarium. The "closed-pimple stage" has certain similarities to the skin spot reported by European investigators, which is attributed to *Oospora pustulans* Owen and Wakefield, and possibly to *Phoma eupyrena* Sacc. and *Spicaria solani* Harz., but the latest information indicates that skin spot, at least in England, is distinct.

CONTROL MEASURES

Affected tubers should not be used for seed purposes in localities with moist, cool climates. Seed treatment will reduce the infection present on the tubers, but will not prevent the infection originating from the soil whenever conditions are favorable for the fungus. (*80*, *107*, *110*, *114*, *141*, *165*.)

RHIZOCTONIA BLACK SCURF OR SOIL ROT

(*Rhizoctonia solani* Kühn; *Corticium vagum* Berk. and Curt.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

In addition to the potato, the fungus *Rhizoctonia solani* attacks a wide range of hosts, affecting primarily the parts below ground or such as are near the soil. In transit or in storage the disease is significant as a decay chiefly in tomatoes, string beans, and turnips. In the potato the fungus induces jelly end rot (pl. 9, C) (see p. 29) and also russet scab and scurf, which frequently are serious enough to affect the marketability of the tubers.

The most prevalent manifestation of the disease in tubers of the potato is the so-called black scurf. This consists of hard, black to brown masses of the fungus, known as sclerotia, which adhere firmly to the skin of the tuber. (Pl. 14, C.) They vary greatly in size and shape. These sclerotia are composed of an extremely close weft of fungus threads and are the form in which the fungus overwinters. They are harmless to the potato in this stage and unless they are present in large numbers do not injure the sale of tubers for table purposes. In cars with high humidity the sclerotia may resume growth, so that the resultant mold affects the price of the lot. The same fungus also attacks tubers, inducing russet scab, and affects other root crops as well. When this occurs the fungus in-

duces death of the superficial layers of the skin. In slight attacks the skin of white varieties becomes russeted. (Pl. 14, D.) In more severe cases deformity of the tubers develops. When the attack by Rhizoctonia is associated with or preceded by insect attack, severe injury to the tuber results. Following flea-beetle injuries (see Scab, p. 43), Rhizoctonia is likely to produce deep cracks and furrows and also to penetrate along the insect punctures. The fungus often invades the holes made by wireworms (see Scab, p. 43), deepening the pits and channels. Rhizoctonia also invades the lenticels, producing small, dry pits.

In attacking the stolons, roots, and main stem of plants, the fungus induces a quite different set of symptoms. There it causes sharply sunken brown lesions of the cortex, that may be so extensive that they girdle the stem or root. If these develop at or near the tender growing tip they frequently destroy it completely. Successively developed sprouts may be destroyed in a similar fashion, so that a cluster or rosette of sprouts without growing tips is formed. If this continues long enough, either very weak, spindling shoots or none at all come through the ground, and poor stands result. If shoots and stolons are attacked less severely or later in their life the plant is not killed, but there is such interference with the movement of food and with other functions that the leaves roll up, wilt, and become discolored. In the potato, small tubers are developed in the axils of the leaves and stems, or the nodes become excessively swollen because of the interrupted movement of food from the leaves to the underground parts. This phase of the disease is known as "little potato" or "all tops and no potatoes."

The fungus itself is so characteristic that its presence aids in the identification of the diseases caused by it. The mycelium is very coarse. At first it is whitish, but later it deepens in color, passing through yellow to deep brown. Frequently the fungus covers or envelops the tissues with a brown mat of its rather coarse strands. When given the opportunity, a colony of this fungus has a radial appearance, the strands resembling densely crowded spokes of a wheel.

More characteristic even than the coarse brown threads of the fungus are its sclerotia or resting bodies. At first they appear as small, soft, white masses; later they become larger and turn dark brown and hard. They usually vary from one-sixteenth to one-eighth of an inch in diameter and sometimes coalesce to form larger masses.

Under very moist, warm conditions in the field, the fungus may develop its spore stage at the base of plants, just at or slightly above the ground level. This stage is known as *Corticium vagum.* The spore-bearing mycelium consists of an ashen-gray network, formation of which is facilitated if a little soil thrown up by rains has formed a film around the stem. The network may extend upward an inch or so. It is so friable that it rubs off readily, and with age it cracks and falls off. Upon alighting under favorable circumstances the spores give rise to new colonies of the fungus. The presence of the collar stage of the fungus is by no means necessarily associated with lesions. Generally the underlying tissues are perfectly sound.

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A. Scab, early stage, mild type
B. Scab, late severe type
C. Black scurf (Rhizoctonia solani)
D. Russet scab (Rhizoctonia solani)

CAUSAL FACTORS

The fruiting stage (*Corticium vagum*) occurs far less frequently than the sterile stage (*Rhizoctonia solani*) of the fungus under discussion. The fungus occurs in soils and lives either on decaying plant trash or on a wide range of both weeds and cultivated plants. For this reason it is known as one of the soil fungi. Experiments in general indicate that the likelihood of getting stem lesions and cutting off sprouts and roots is greater when untreated tubers bearing sclerotia are planted than when sclerotia-free tubers or treated tubers bearing sclerotia are used. This indicates that not only the fungus in the soil, but probably even more the fungus that arises from germination of the sclerotia, is responsible for the disease. The spores play a minor rôle in the propagation and distribution of the fungus.

Field observations and some experimental work with temperatures indicate that both soil moisture and temperature determine the occurrence and pathogenicity of Rhizoctonia. The fact that the spore stage of the fungus does not develop unless there is rainy, foggy weather, or unless the plants shade the ground very effectively and cause a blanket of wet air, is further evidence that air with an abundance of moisture favors the organism.

Infection in the soil itself, when there is a moderate water content, seems to be determined by several factors. One is the presence of virulent strains. There seem to be a great number of strains of Rhizoctonia, many of which are not highly pathogenic. Given a virulent strain and a susceptible host, the temperature of the soil seems to be the most important limiting factor for the development of the fungus as well as for the appearance and severity of the disease. Field observations indicate that temperatures below 70° F. are more favorable for the development of the fungus than higher ones. Frequently the stems and roots of plants that show no strands or sclerotia of the fungus during the summer become literally covered with them at the onset of cooler weather. The disease is more severe in cool seasons and sections than in warmer ones.

Experimental work with the potato substantiates these observations. The fungus produced lesions in potato stems and roots at temperatures ranging from 46° to 80° F. The greatest damage was done between 59° and 70°, though even at 46° severe damage resulted. There was no damage whatsoever at 86°.

CONTROL MEASURES

Very little can be done in the field to control the disease. The avoidance of overcrowding and the drenching of the soil tend to minimize damping off and crown injury.

Field injury of the underground parts is difficult to control. In the case of the potato beneficial results are reported from some sections in certain seasons when scurf-free potatoes are planted or when tubers bearing sclerotia are treated with mercuric chloride 1:1,000 (4 ounces to 30 gallons of water for one and a half hours), or with hot (125° F.) formaldehyde solution (1 pint to 15 gallons for four minutes). Good results have also been obtained with certain organic mercury compounds used in solution or applied at the rate of 2 ounces per bushel to presprinkled seed. (*45*, *109*, *117*, *132*, *139*, *140*, *145*, *147*, *148*, *149*, *162*, *163*, *164*.)

RHIZOPUS SOFT ROTS

(*Rhizopus* spp., principally *R. nigricans* Ehrenb. ex Fr.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Rhizopus soft rot (also known as leak and affected tubers as leakers and melters) has been observed most frequently in California, Idaho, and Washington, both in the field and in transit and storage. The characteristic symptom is disintegration of the tuber tissues into a soft leaky mass, from which a brownish-yellow liquid drops when the tissues are under pressure or when they are freshly cut. The rot begins as a brown discoloration and a water-soaked area about a wound, which soon enlarges rapidly and involves the entire tuber, the cortical tissues resisting the rot the longest. The exterior of the tuber with advanced rot is brown and the interior a dirty white, which turns brown upon exposure to the air. (Pl. 12, C, D.) Generally it is not possible to differentiate this rot from leak caused by Pythium species (see Leak, p. 33) unless the fungus is identified microscopically or culturally. Unlike the situation in many other vegetables, even under favorable temperature and moisture conditions, there usually is no external sign of the coarse, white, stringy mycelium of the fungus with its white, glistening heads, or sporangia, which later turn brown or black as the myriads of spores which they contain become mature.

This decay has been experimentally produced in 16 species of fresh vegetable crops in addition to the potato.

CAUSAL FACTORS

Rhizopus rot of the potato may be induced by 10 species of Rhizopus: *R. nigricans* Ehrenb. ex Fr.; *R. reflexus* Bain; *R. artocarpi* Racib.; *R. arrhizus* Fischer; *R. nodosus* Namysl.; *R. delemar* (Boid) Wehmer and Hanzawa; *R. tritici* Saito; *R. maydis*, Bruderl; *R. oryzae* Went and Pr. Geerligs; and *R. chinensis* Saito.

Species of Rhizopus seem to be everywhere present, in the soil in the form of mycelium and spores, and in the air in the form of spores. As a result plant tissues may be contaminated with them anywhere in the field, during the harvest, in the packing process, in containers, in transit, and in storage. However, the amount of inoculum varies from place to place. Some soils and fields are more heavily infested than others. The air in a storage house that is not kept clean is likely to carry more spores than the outside air. Potatoes from any section, therefore, may develop soft rot if mechanical injuries are present and temperature and moisture conditions are favorable for the development of Rhizopus species. Temperature together with relative abundance of spores or mycelium also determines which of a given lot of species of Rhizopus will attack a particular tissue.

The Rhizopus species that may cause tuber rot can be arranged in three groups on the basis of their optimum temperatures. The optimum temperature for *R. nigricans* and *R. reflexus* is between 68° and 71° F.; for all the others except *R. chinensis* it is about 86°. The optimum for *R. chinensis* is about 95°.

In spite of the fact that the organisms of the middle group cause a higher percentage of infection and more severe decay under experimental conditions, it has been demonstrated that *Rhizopus nigricans*

is most commonly found in rotting vegetables, *R. tritici* ranking second. *R. nigricans*, which induces most of the rot, makes only slight growth below 44° F., grows best between 77° and 82°, and is checked by temperatures above 90°. Although wounds are essential for invasion, it appears that fresh, unwilted, or turgid tissues are most susceptible. Rhizopus rot is not a secondary invader of lesions to the extent that bacterial soft rot is.

The combination of a soil heavily infested with Rhizopus, of high temperatures of soil and air, and the harvesting of tubers with forks that cause many wounds is probably responsible for the high incidence of this disease in potatoes in the Stockton, Calif., district.

CONTROL MEASURES

Since the spores of the fungus are practically everywhere present, control of Rhizopus rot is not an easy matter. General sanitary measures are recommended for the field and for the packing and storage house. All possible care should be exercised to avoid breaking the protective skin; also all precautions possible should be taken to keep the plant surfaces dry. In storage and in transit, precautions should be taken against the accumulation of moisture in the air, and temperatures should be kept low. The lower the temperature the less likely it is that rot will occur and the more its rate of development will be retarded. (*61*, *63*, *83*, *125*, *126*, *182*.)

ROOT KNOT

(*Heterodera marioni* (Cornu) Goodey)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Root knot, root gall, or big root is characterized by galls on the underground parts of affected plants. In many vegetable crops the roots are the only parts affected, but in the potato the disease is marked by roughened, discolored, and warty surfaces on the tubers in addition to small galls on the rootlets. On tuber and root crops the galls are of direct significance in the market because they reduce the quality of affected stock, rendering it unsightly for table use and impairing its storage qualities. Affected tubers are entirely unsuited for seed purposes because they carry the disease into uninfested areas.

On plants other than root crops the disease is of only indirect market significance, but in these and in tuber crops it is very important from the point of view of production. The galls interfere with the absorbing and conducting functions of the roots, so that the plants become deficient in water and soil nutrients. They also use up a great deal of the food made by the leaves. As a result, affected plants languish and often are decidedly stunted and yellow. In leafy crops this condition expresses itself directly in the edible part of the plant. In plants such as muskmelons (cantaloupes) and tomatoes a reduced setting and poorly flavored fruits that tend to deteriorate rapidly are the results.

The disease is common in greenhouses everywhere and occurs in all parts of the United States except the most northern States.

The Atlantic and Gulf coastal regions are most seriously affected, and sandy soils are more seriously infested than heavy ones.

The eelworm or nematode (*Heterodera marioni*) attacks about 500 wild or cultivated plants. Among truck crops the following are reported as more or less susceptible: Asparagus, bean, beet, cantaloupe, carrot, celery, cucumber, dasheen, eggplant, garden pea, lettuce, muskmelon, okra, onion, pepper, potato, salsify, spinach, strawberry, tomato, and watermelon.

Since this disease directly affects only the underground parts, in case of a mild attack plants may show no signs of disease above ground except possibly a slight dwarfing. Plants that are badly infested, however, are noticeably smaller and paler green than normal ones. Such plants wilt readily in dry, hot weather, because their root systems are reduced so much that they can not obtain sufficient moisture.

The knots or galls may vary considerably in size and shape, depending upon the number of nematodes present. On tubers and fleshy roots the disease occurs as small pimplelike swellings or larger protuberances of the surface, which ordinarily become roughened at the infected places. When these swellings are some distance apart they are circular, but if they occur close together they take on various shapes and sizes, causing decided malformations. (Pl. 15, C.)

Eelworm-infested tubers and roots are sometimes confused with those affected by other diseases—crown gall of beets, clubroot of crucifers, and "pimply potatoes" due to flea-beetle injury (see p. 44)—which also involve a swelling of the diseased tissues. The nematode galls, however, may usually be readily distinguished from other maladies by the presence of small, white, pear-shaped nematodes, the adult females, that may barely be seen with the unaided eye when well-infested tissues are broken or teased apart.

CAUSAL FACTORS

The organism that induces root knot is a nematode or eelworm, *Heterodera marioni*. This eelworm occurs widely in most of the older trucking sections of the southern portions of the United States and in greenhouse soils everywhere. The eelworms and the eggs live over in the infested plant débris or in the soil. Each female may lay more than 500 eggs which will develop into the eelworm stage. In warm regions it is possible for nematodes to pass through 10 to 12 generations in one year. At temperatures below 55° F. the nematodes are inactive and do little damage. For this reason it is possible to raise some crops such as lettuce and celery on nematode-infested soils in the warmer sections of the United States in the winter. Larval and adult male worms as well as larval and immature females can move through the soil and plant tissues. When they come to the roots of a suitable plant they bore their way into it, especially just back of the growing points of the fine rootlets. Once within the tissues, the eelworms begin to enlarge and absorb nourishment. The female enlarges enormously in diameter so that she eventually becomes pear-shaped and large enough to be visible to the unaided eye (one fortieth to one twenty-fifth of an inch), while the male remains slender and small. The presence of the worms

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A, B. Flea-beetle injury
C. Nematode injury
D, E. Tuber-moth injury

causes an irritation of the tissues, which results in increase in size of the affected organs. If no hosts are available when the eggs hatch, the young worms promptly die. Unfortunately, however, they can attack practically all vegetables and many other plants. Certain cereals, such as rye, are immune.

The parasite is easily carried about on the roots of plants. It is therefore particularly important that it be kept out of seed beds. Another reason is that plants have a better chance of overcoming or tolerating the disease if they are not infested in their early stages of development.

CONTROL MEASURES

Control of the parasite is a difficult matter. When relatively small quantities of valuable soil are involved, as in greenhouses or in seed beds, it pays to sterilize the soil with steam or to apply boiling water at the rate of 6 or 7 gallons per cubic foot of soil. In the field, especially in hot, dry seasons, summer fallowing is very beneficial, because it starves out the worms. On small garden plots in the South it is advisable to plant grain the first year, to let them be chicken yards the next year, and gardens the third year. In Florida it has been found that when calcium cyanamide is thoroughly mixed into the soil at the rate of 1,000 pounds or more per acre, and the soil then irrigated or wetted down, the number of nematodes is sufficiently reduced to permit a susceptible crop to be grown. With cowpeas, soybeans, and some other crops the most effective control is the use of resistant varieties.

Extreme care should be used to prevent the transportation of infested plants into noninfested or new sections. For this reason there are definite quarantine regulations against this disease in several States. (*24*, *48*, *152*.)

SCAB AND INJURIES RESEMBLING SCAB

(*Actinomyces scabies* (Thax.) Güssow, insects, and animal depredators)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Scab has been reported from practically every potato-growing section of the United States. In some seasons it is the most serious potato disease for some sections. It attacks only the underground parts of the plant, the effects being most serious on the tubers. From the point of view of yield the disease is of little consequence, but from the point of view of production of marketable tubers it is extremely important. The economic importance of scab lies in the fact that it renders tubers unsightly, thus lowering or destroying their market value. The disease apparently does not affect the vigor of the growing plant or the total yield, although it sometimes attacks the underground portion of the stem and the roots, as well as the growing tubers.

Scab is characterized by rough, corky elevations, or by pits on the surface of the tuber. If tubers are affected early or severely, the lesions are deep or pitted (pl. 14, B); if late, they may be shallow and superficial (pl. 14, A). In the early stages the lesions are minute and reddish or brownish in color and may be raised. (Pl. 14, A.) In response to stimulation involved in destruction of the cork layer

by the fungus, new layers of cork are developed underneath, which lead to the death of the tissues cut off. If these layers of cork are deep, the pitted or deep type of scab results. Often the lesions coalesce, and in severe cases the entire tuber may be covered with a rough incrustation. Tender, white-skinned varieties appear to be more susceptible than rough-skinned russet varieties.

The larva of the flea beetle *Epitrix cucumeris* Harris causes injury to the potato tuber which may in some cases resemble the early stages of common scab. If the pimples or furrows caused by the flea beetle are cut through, peculiar tough splinters of corky tissue are found extending perpendicularly into the tuber. These splinters may be one-fourth of an inch in length. They are characteristic of this type of injury and distinguish it from common scab, nematode injury, grub injury, or wireworm injury. (Pl. 15, A, B.) In the simplest cases flea-beetle injury is marked by corky pimples, which may be broken open. In more advanced stages the surface of the tuber is a mass of pimples and furrows or channels formed by the breaking of the skin. The margins of such furrows frequently are elevated and swollen, so that they stand above the surface of the tuber. This type of injury is known as worm tracks. Flea-beetle injury is most common in Colorado potatoes. It injures their appearance and generally necessitates very deep paring. The furrows and punctures frequently become infected with Rhizoctonia, which deepens them.

Wireworm injury is caused by the larvæ of several species of hard-shelled beetles (Elateridae). It consists of channels through the tuber skin and into the flesh of the tuber. The former type occasionally is mistaken for scab. These channels vary with the size of the wireworm, approximately one-sixteenth of an inch in diameter. In the interior of the tuber there is a brown discoloration surrounding the bore of the tunnel. The tunnels are often well walled off with cork, but frequently decay organisms get started in them. In tuber-moth injury, which is caused by the larva of the tuber moth (*Phythorimoea operculella* Zeller), the channels are mostly shallow and often lead to collapse of the corky layer. The tunnels usually begin at the eyes, where sawdustlike heaps of material give telling evidence of the insect's activities. (Pl. 15, D, E.) Tuber-moth injury occurs mostly in tubers from southern California. Wireworm injury may appear in tubers from any section. It is often severe in tubers that are grown immediately following sod.

Grub and rodent injuries consist of large cavities. Unlike the lesions of common scab, the cavities are not walled off with flaky, rough cork layers. Cavities caused by white grubs (larvæ of *Phyllophaga* spp.) usually have more or less smooth surfaces, while those made by rodents are definitely ridged and furrowed. The injuries caused by fowls and other birds are characterized by definite peck marks.

CAUSAL FACTORS

Actinomyces scabies is by some considered a bacterial form and by others a fungus. It is a soil inhabitant. It may overwinter in the soil or on tubers in storage and proceeds from either of these sources to the tender-growing underground stems of the potato. The young tubers are especially susceptible, although the main stem

of the unthickened stolons also becomes attacked when conditions are favorable for the fungus. The organism does not attack mature tubers, and as a consequence the disease does not progress nor spread after the harvest.

It is a general observation that scab is more severe in neutral and alkaline than in acid soils. The organism grows best and causes most severe scab when the soil reaction lies between pH 5.3 and 8.5. Consequently, if soils are acid enough scab does not develop, even when untreated scabby tubers are planted. It has also been noted that scab is more severe in those sections having high temperatures during the growing season and that it is most severe in a given section during a warm season. The fungus itself grows better at high than at low temperatures. It does not grow at all below 46° F., grows best between 78° and 86°, and ceases growth at 100°. Temperatures of the soil ranging between 68° and 82° are most favorable for scab development. Scab is generally most severe in soils whose moisture condition is favorable for rapid tuber development. It is retarded by very dry soil, and since the organism is a strong aerobe, it does not develop in soils with excessive moisture.

CONTROL

In some sections it is found practicable to treat slightly alkaline soils with sulphur so that they become slightly acid. In other sections this treatment has not been found satisfactory. The efficiency of the sulphur is sometimes increased if it is inoculated with certain species of bacteria that oxidize the sulphur into acid compounds. Since the fungus can be carried into the field on infected tubers, it is not advisable to plant badly scabbed tubers. Slightly scabbed tubers may be planted if they are previously treated with either formaldehyde solution or corrosive sublimate solution. The standard method of formaldehyde treatment consists in soaking the tubers for 2 hours in a solution made of 1 pint of commercial (40 per cent) formaldehyde in 30 gallons of water. A modification consists in shortening the treatment from 2 hours to 30 minutes if the tubers are presoaked in water. If the potatoes are covered with burlap after the soaking, the efficiency of the treatment is increased. A final modification consists in using a solution of double strength at a temperature of 122° F., the time being reduced to 2½ minutes. The standard corrosive sublimate treatment consists in soaking tubers for 1½ hours in a solution of 1 part by weight of corrosive sublimate in 1,000 parts of water, or 4 ounces of corrosive sublimate in 30 gallons of water. It is found by some that the time of soaking can be shortened to 30 minutes. Both treatments can be made more effective if the tubers are sprinkled with water and covered with a tarpaulin for 24 to 36 hours previous to soaking.

Whenever possible green manuring should be practiced. It is considered that the beneficial results of this procedure are not due to increase in the acidity of the soil but to the stimulation of growth of other soil organisms that are antagonistic to the scab organism. Unless the soil is rather acid, wood ashes, lime, and stable manure should not be used on soil immediately preceding a potato crop. Finally, if the soil is inclined to produce scabby potatoes, it is advisable to plant only the more scab-resistant russet varieties. (*5*, *58*, *75*, *94*, *95*, *96*, *100*, *102*, *153*, *154*, *181*.)

SCALD

It is a general observation that tubers exposed to the intense sunlight of a hot day immediately after digging, or allowed to be on or near the surface of the soil after digging on hot days, often develop breakdown of the outer or even of the deep-seated tissues. This condition is known as scald. It is not known to what extent it is due to the direct killing effect of certain parts of the sun's rays, to coagulation of the protoplasm owing to high temperatures, or to indirect effects of disturbances in the respiration of the affected tissues, all of which can produce death of the tissues. It seems quite clear, however, that direct action of the sun's rays on the tissues is not necessary to produce scald. Affected tubers are unmarketable and are a menace to healthy ones, because the killed tissues are readily invaded by rot-producing organisms.

Scald may appear first as large blisters, which soon become sharply depressed (pl. 12, A), or as slightly bleached areas with a very irregular, lobed margin of a darker metallic color. Upon cutting into such areas the tissues are found to be somewhat soft, watery, and only slightly discolored, or not discolored at first. If discoloration sets in, it is first gray, later brown, and finally black (pl. 12, B), as in black-heart. (See Black-heart, p. 10.) Bluish-black patches often surround the lenticels.

The killed tissues may be set off from the uninjured by a brown, irregular line. If the killed tissues dry out into a brittle or tough sticky mass, and the extreme discolorations do not develop, it is frequently difficult to determine whether freezing or scalding killed the tissues. Sclerotium rot, leak, and Fusarium rot often produce the interior discoloration associated with scald. These rots also are frequent secondary consequences of scald. If the killed tissues dry out before infection takes place, they become tough and leathery before discoloration sets in. Unfortunately, no way has yet been devised for determining whether tubers have been scalded. Several hours to several days may elapse after injury takes place before the first symptoms appear.

It is not advisable to ship potatoes showing scald, because of their extreme predisposition to bacterial soft rot and to other decays. (*167, p. 32.*)

SCLEROTIUM ROT (SOUTHERN BLIGHT)

(*Sclerotium rolfsii* Sacc.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Sclerotium rot is essentially a disease of truck crops of the southern part of the United States. It affects not only the potato but many other vegetables as well. The disease is of considerable importance in transit; on roots of beet, carrot, and sweetpotato; on asparagus stems, and potato tubers; on bulbs, such as onion and garlic; on fleshy buds, such as cabbage and head lettuce; and on the fruits, bean, pepper, tomato, muskmelon, squash, and watermelon. The most serious losses in transit occur on southern potatoes and tomatoes.

The parasite is one of the soil fungi, and when moisture and temperature conditions are favorable it can become parasitic on a

wide range of hosts. It attacks growing plants most frequently at the crown or foot, from which it spreads through the entire underground system, producing wilting and death. Attacks of the fungus, however, are not restricted to the crown of young or growing plants. Fleshy storage and fruit organs are attacked when they lie on infested soil or become spattered with it.

Sclerotium rot is a southern disease. The fungus seems to prefer sandy or loamy soils, and in such soils the disease is likely to appear during or following wet spells. Its geographical occurrence is largely determined by temperature, growth being very poor below 68° F. On the market, Sclerotium rot is found most abundantly in southern potatoes shipped during May, June, and July. During these months it sometimes ranks with Fusarium rot and bacterial soft rot as a serious potato disease.

The character of the rot of fleshy organs depends in part upon the kind of vegetable involved and upon external conditions. In some vegetables the fungus may produce an exceedingly wet rot—a condition known as "melter" in potatoes and muskmelons; in others, such as the sweetpotato, squash, and watermelon, the rot is always dry; in potatoes and muskmelons it is dry if the surrounding air is dry.

There is no uniformity in the color of the rot. In some cases, as in potato rot, so little discoloration results that the name "white rot" has been used. In cabbage, on the other hand, the affected tissues are black, and in the sweetpotato they are brown.

In the early stages the rot on potatoes is a white and practically odorless decay; in the later stages it takes on a yellow color. (Pl. 4, F.) In mild cases there are only slightly sunken spots, the soft, decaying material of which can be separated easily from the healthy by pressure. Often, however, the decay penetrates deeply into the tissues by a narrow channel and then enlarges until it involves the entire tuber, producing the melter stage. (Pl. 4, F.)

Differentiation of this rot from bacterial soft rot is not always easy macroscopically. The latter, however, in nature generally is marked by a vile odor. When the fungus has grown out on the surface of the lesions diagnosis is easy and generally specific.

As in several other diseases induced by general parasites, the fungus itself is so characteristic when mature that it is not readily confused with any other and can be used for diagnostic purposes. It has a moderately fine, silky, white mycelium which frequently grows abundantly on the surface of some hosts. On flat surfaces the strands of the fungus grow in a more or less linear radial fashion, forming fans. (Pl. 4, G.) The grouping of individual hyphæ or filaments into strands which advance as units sometimes makes the mycelium appear coarse. These fans are frequently found on the soil, on plant organs, and even on crates and bags. As the fungus gets older, it produces small fruiting bodies, sclerotia (pl. 4, G), which are at first whitish, but which later resemble radish seed in color, shape, and generally in size. Their size, however, depends somewhat upon the tissues on which the fungus feeds. On most vegetables the sclerotia are about the size of mustard seed.

In the field, the young leaves of affected plants first show drooping during the heat of the day and recover at night. Wilting progresses,

however, and after three or four days the foliage yellows, and complete wilting sets in. The leaves die, the stem loses its color, dries out, and dies. At the crown of such plants, one-half to 1 inch below the ground level, shrunken, discolored areas are found. These areas are small, brown, and firm at first. Later they enlarge and, though firm, become water soaked. Finally, the lesion at the crown becomes covered with the fungus, which produces its sclerotia in abundance.

CAUSAL FACTORS

In the field primary infection by the fungus *Sclerotium rolfsii* proceeds from the soil. Under conditions favorable for the fungus its mycelium develops from the sclerotia, in which form the fungus probably overwinters. Apparently it spreads entirely by mycelium, both in the field and in storage and transit, because, so far as known, the organism does not produce spores.

As a rule the fungus at first attacks only young, tender parts of plants in the field. It can attack plant tissues through the unbroken skin, but wounds naturally facilitate its entry and establishment. Growth on host tissues seems to increase the pathogenicity of the fungus, so that it can infect other tissues without wounds. Moisture and temperature are the most important limiting factors for development of the fungus and of infection. The minimum temperature for growth of the fungus is about 46° F.; it develops most rapidly between 86° and 95°, and generally stops growing at 98° to 104°. Since the fungus grows slowly at temperatures below 68°, an incubation period of appreciable length is likely at these temperatures, and it is possible, therefore, to pack and load apparently sound but contaminated vegetables, which under conditions of higher temperatures may develop lesions later on. The incubation period for potato tubers has been reported to be about six days, and at about 70° rot lesions an inch in diameter are produced in about 15 days.

CONTROL MEASURES

Control of Sclerotium rot in warm sections is difficult, like that of other diseases induced by soil fungi. The fungus can grow on a wide range of crops and ornamental and weed plants. Deep burial of the fungus in the soil seems to kill or weaken it. Accordingly, deep plowing that buries the mycelium or sclerotia 5 inches or more seems to decrease the amount of the disease.

Avoidance of harvesting and packing operations during wet weather and drying wet plant surfaces before packing and loading are advisable precautionary methods. (*35*, *60*, *64*, *176*.)

SILVER SCURF

(*Spondylocladium atrovirens* Harz.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

The disease known as silver scurf is very generally distributed over the United States. Its only economic significance is that it disfigures tubers somewhat and in severe attacks leads to excessive drying out.

The fungus kills the superficial layers of cells of the tuber. The affected cells take on a silvery appearance which is most readily seen when the tubers are wet. These areas may be small or large, sometimes involving almost the entire surface of the tuber. (Pl. 3, E.) The injury becomes more pronounced in storage, especially under moist conditions, which favor the formation of a superficial sooty layer composed of the fungus. Badly affected tubers shrivel more rapidly than sound ones.

CAUSAL FACTORS

The fungus (*Spondylocladium atrovirens*) that induces silver scurf lives in the soil, from which infection of the tubers proceeds. It also lives over in tubers and can be carried into the field on them. There is no spread of the fungus from tuber to tuber, although there is development of the fungus under favorable conditions in transit and storage.

The fungus requires a good supply of moisture for best development. It does not grow at temperatures below 35° F. nor above 86°, and grows best at temperatures between 70° and 81°. No data relative to the temperature and moisture requirements for infection are available.

CONTROL MEASURES

Seed treatment that is effective for scab control is not sufficient to kill the silver-scurf parasite. No method of killing the fungus in the soil is known. The only practicable control measures are avoidance of planting affected tubers and the practice of long rotations. (*106*, *155*.)

SPINDLE TUBER

The disease now called spindle tuber has been known for several years as "running out" or "off shape," but recently it has been found to be one of the virus diseases. It has been observed in potatoes in the East as well as in the West. Some varieties have shown as high as 95 per cent infection, and in cases of severe infection the yield may be reduced as much as 50 per cent.

The leaves of diseased plants are smaller and darker green than normal, and the margins are sometimes rolled, while the stems are more or less spindling and upright. Spindle tuber is not only an important disease from the point of view of production, on account of the reduction in yield, but is also important from the market point of view on account of the poorly shaped tubers produced by affected plants. Affected tubers are characteristically spindle-shaped or cylindrical, the ends being abnormally long and pointed. (Pl. 3, D.) This seriously affects their grade as table stock. Since the virus is carried in the tubers, no affected stock should be used for seed purposes.

Spindle tuber is spread in the fields chiefly through the agency of such insects as aphids and grasshoppers. There is also evidence to show that the disease may be transmitted by cutting knives during the preparation of seed.

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Control of this disease is best obtained by careful selection of seed stock and roguing of undesirable plants as fast as they appear during the growing season or the use of certified seed. (*37*, *52*, *53*, *156*, *159*, *188*.)

SURFACE MOLDS

Under transit or storage conditions potatoes often show a superficial growth of mold. This does not injure the tubers except as it affects their appearance. Occasionally the fungi penetrate and cause slight decay. Various fungi are involved, including brightly colored ones. The most common ones are *Fusarium* sp., *Rhizoctonia* sp., and *Stysanus stemonitis* Cda. The growth of these fungi is favored by condensation of moisture on the tubers and by the presence of juice from near-by frozen or rotting tubers. The growth often develops on wet, dirty tubers and on cut or bruised areas. The presence of the mold is objectionable in shipments because it gives the lot a bad appearance. If care is taken not to load or store dirty, wet tubers and to provide aeration in storage and in transit, the growth of surface mold can be avoided.

VERTICILLIUM WILT

(*Verticillium albo-atrum* Reinke and Berth.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

The symptoms of Verticillium wilt are very similar to those produced by *Fusarium oxysporum*. (Pl. 9, D.) (See Fusarium Wilts, p. 25.) The disease occurs in the cooler portions of the United States. Its economic importance lies in the fact that it kills growing plants, thus reducing the yield, and causes discoloration of the vascular elements of tubers, thus lowering their market value and rendering them unfit for sale as seed. Verticillium wilt also occurs in okra, eggplant, and other plants.

The first evidence of the disease in the growing plant is lack of turgor in the portions of the leaves farthest from the veins. The lower leaves are affected first and after wilting drop off one at a time. Frequently plants are stunted and do not die for a long time. If the stems or petioles of affected plants are cut longitudinally or crosswise, the vascular ring is found to be brown to black, the discoloration extending from the smallest rootlets through the stem into the petioles. There does not seem to be any breaking down of tissues such as occurs in the Fusarium wilts at times, but the wilting is attributed to obstruction of the water vessels. At times a grayish mold, which enables one to distinguish the disease from Fusarium wilt, appears on the wilted stems at the ground level. In the potato the discoloration extends from the plant through the stolon into the tuber. So far as known, the fungus does not cause decay of the tuber tissues.

CAUSAL FACTORS

The fungus *Verticillium albo-atrum*, which induces this disease, is one of the soil fungi. Infection of the growing plant can proceed either from infected seed pieces produced by diseased plants the

previous season or from the soil. Infection also spreads in the field from plant to plant. Tubers become infected from plants through the stolons.

There is no development or spread of the disease in transit or in storage. The distribution of the disease indicates that moderate temperatures favor the organism, at least that the disease does not develop in extremely high temperatures. No detailed data relative to temperature or moisture requirements of the fungus and the disease are available.

CONTROL MEASURES

Control involves avoidance of planting diseased tubers and the practice of crop rotation and the use of certified seed. To avoid diseased tubers, the best method is to rogue the diseased plants in a seed plot. Absence of stem-end discolorations does not guarantee absence of the fungus, and, on the other hand, discoloration does not necessarily mean presence of this fungus any more than it does the presence of Fusarium species. It has been found in Oregon that under the conditions existing there a 5-year rotation will eliminate the fungus from the soil. (*97*, *127*, *136*.)

VIOLET ROOT ROT

Rhizoctonia crocorum (P. ex Fr.)

Violet root rot affects the roots and underground parts of a wide range of plants. It is of direct significance from the market point of view only in root or tuber crops, on which it produces lesions that blemish the organ or open the way for secondary parasites.

The parasite itself furnishes the most characteristic diagnostic features. Its violet-brown mycelial strands cover the surface of the tuber or other affected organs with a dense mantle or weft. Sometimes, however, the mycelium is sparse and diffuse. It bears minute aggregations, microsclerotia, which are much darker in color than the mycelium. Under very wet conditions the fungus grows above the ground on the stem of the affected plant, at times extending onto the leaves.

The fungus produces on the tubers lesions that tend to be most abundant at the stem end. The lesions are at first small and violet in color; later they may enlarge and increase in number until the entire tuber is involved. Shortly after a lesion develops, the epidermis breaks and exposes the cortical tissue, which has an ashen-gray color. The cells immediately below are purple, and below these is a zone of water-soaked cells. The exposed tissues become dry and powdery. At times, especially in plants other than the potato, enough roots are affected by the fungus to produce yellowing and wilting in the aboveground parts.

The parasite is one of the soil fungi and generally overwinters in the form of sclerotia. The mycelium grows through the soil and at times even on the soil. It is favored by wet weather.

Control involves the use of seed from noninfested soils, seed treatment as for scab, and crop rotation. (*36*, *132*.)

WART

(*Synchytrium endobioticum* (Schilbersky) Percival)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Potato wart became known in the United States in 1918, and its discovery caused considerable apprehension as to the future of the potato industry. As a result of rigorous quarantine measures, including the growing of prescribed immune varieties, and the fact that there is no movement of potatoes from the infested areas, the disease is limited to certain counties in Pennsylvania, West Virginia, and Maryland, into which it was introduced by tubers shipped from Europe. The chief economic significance of the disease is the fact that it causes warts on the tubers which either lead to their complete destruction or so affect their appearance that they become unsightly and unmarketable.

Although the fungus may attack all parts of the plant, the most severe effects are produced on the tubers. It enters the eyes and causes abnormal growth that is warty and cauliflowerlike. (Pl. 13, A, B.) If infection takes place early, the entire tuber may be converted into a worthless, spongy mass. Similar warts may appear on stolons and other underground parts. Young warts usually have the same color as the affected tissues, later turning light brown and finally dark brown or black. Black color generally is associated with decay, which almost invariably sets in.

CAUSAL FACTORS

The fungus *Synchytrium endobioticum*, which induces wart, can live in the soil for many years. It may be carried on tubers grown in infested soil. Infection proceeds from the soil or from warts on tubers used for seed. There is no development or spread of the disease in transit or in storage.

The fungus requires a moist soil to produce infection. It is able to cause infection throughout the temperature range of the potato plant, but is most favored by temperatures between 59° and 64° F. There is a very decided difference in the susceptibility of varieties to its attack. Some of the best American varieties, such as Irish Cobbler, Green Mountain, and Spaulding Rose, are entirely immune. Unfortunately, the Rural type, which is a very important one in central United States, has not yet furnished an immune variety. Some irregularities and protuberances are sometimes formed on supposedly immune varieties, but there is no serious development of the disease.

CONTROL MEASURES

There is no practicable way of eradicating the fungus from the soil. Steam heat and various chemicals have been used with success, but these are impracticable on a large scale because of the cost and injury to the soil. For this reason a very strict quarantine has been maintained about the infested regions. The most effective control measure is the use of wart-immune varieties. Resting spores on the surface of tubers are not killed by ordinary seed-potato disinfection. They can resist dry heat for six or seven days at 140° F., but are killed in moist heat at 160° within five minutes. (*28*, *46*, *47*, *57*, *69*, *124*, *183*, *184*, *185*, *187*.)

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